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THE ETIOLOGY OF ARTHRITIS DEFORMANS IN CHILDREN *

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IOWA CITY

That the deforming types of arthritis have been existing from earliest historic times can scarcely be doubted. In mummies exhumed from Roman tombs, the disease has been definitely recognized, as is, for example, described by Moore.[1] In the treatise of Sudhoff,[2] however, the declaration is made that the disease was extraordinarily common in Egypt and also among Germanic tribes along the Baltic, probably because of the dampness of the climate. In England, gout and chronic joint conditions have long been almost endemic, although the former is far less common now-a-days. On the continent, however, there was formerly less mention of the disease and Trousseau[3] commented on its rarity. At present the disease is usually encountered sporadically, although Beek[4] found a region in Siberia where from 6 to 46 per cent. of the population, chiefly children between 8 and 13 years, have a chronic osteoarthritis.

When one attempts to find reference to the disease in children in the early medical literature, one is struck with the almost complete lack of data on this subject. Whether this is owing to the fact that the disease did not exist in the first decade of life, which is unlikely, or whether such joint diseases were not carefully differentiated, cannot be stated definitely. Deforming arthritis does not receive mention in the Kinderbuch of Felix Wurtz,[5] although reference is made to other joint affections. Meissner,[6] in his pediatric bibliography with its list of titles of papers published previous to 1850, was also silent on

* From the Department of Pediatrics, College of Medicine, State University of Iowa.

1. Moore: Rheumatic Arthritis from a Roman Tomb, Tr. Path. Soc. London **34**:286, 1882-1883.

2. Sudhoff, K.: Translated by Stockman, F. J. Ann. Med. History **1**:111, 1917.

3. Trousseau: Clinical Medicine: New Sydenham Society **4**:408, 1871.

4. Beek, E.: Ueber Osteoarthritis Deformans, Arch. f. Klin. Chir. **86**:662 (March) 1918.

5. Wurtz, F.: Translated by Fox, A. L.: An Experimental Treatise in Surgery, 1656.

6. Meissner, F. L.: Grundlage der Literatur der Pediatrik, Leipzig, 1850.

this subject. The first case in children is supposed to have been recorded by Cornil[7] in 1864, and after him Charcot[8] was able to refer to two cases in children less than 12 years of age. Barthez and Rilliet[9] stated that gouty rheumatism was of such rarity in childhood that it might conveniently be passed over in silence. In the monograph of Moncorvo[10] we find the first adequate treatment of the subject, although in the historical discussion this author is struck with the dearth of literature, and in correspondence with his teacher, Bouchut, was informed that the latter had not seen six cases in twenty years, wherefore, he made no reference to it in the fifth edition of his work. He also quoted Niemeyer (1869) who stated that infants enjoyed an absolute immunity from nodular rheumatism, one of the terms commonly applied to the disease under discussion. The difficulty at that time and previously in distinguishing the various types of hyperplastic joint disease in children, owing to the lack of the finer diagnostic tests, led to confusion on the subject, and this confusion, similar to that existing in the classification of the disease in adults, was mirrored in the terminology. Thus, while Charcot's[11] rheumatisme noueux was used by many, one encountered also the term "benign hyperplastic synovitis" in the article by Von Wahl[12] in Gerhardt's Handbuch, and rheumatoid arthritis of English authors. Von Wahl stated that this disease was extremely rare in childhood and had been seen by him only following the acute polyarthritis of infectious diseases. In this country, reference was made by Louis Smith[13] to a case of arthritis deformans seen in 1871, and he spoke of it as being "one of great rarity." Twelve cases were collected by Lacaze-Dori[14] and a few of the more striking facts were mentioned, such as the self-limited nature of the affection and the better prognosis in children than in adults. Chlorosis, humidity and cold were considered as contributory etiologic factors. In his opinion, a cure took place after a period of about one year. From that time on occasional references to the disease appeared in the literature.

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7. Cornile: Memoire sur les Coincidences Pathologiques du Rheumatisme Articulaire Chronique, Mem. d. l. Soc. d. Biol. **1**:3, 1864 (Fourth Series).

8. Charcot: Lecon Cliniques sur les Maladies des Viellards et les Maladies Chroniques (1866), Paris **8**:262, 1890.

9. Barthez and Rilliet: Maladies des Enfants **2**:114, 1866.

10. Moncorvo: Estudo Sobre ö Rheumatismo Chronico Nodosa na Enfancia e seu Tractamento a Proposito de una Caso Observado em ema Menina de 2 annos. etc., Rio De Janeiro, 1879.

11. Charcot: See reference No. 8.

12. Von Wahl: Gerhardt's Handbuch der Kinderkrankheiten **6**:416, 1880.

13. Smith, L.: A Treatise on the Diseases of Infancy and Childhood, 1876, Ed. 3, p. 309.

14. Lacaze-Dori: Etude Clinique sur la Rhumatisme Noueux Chez Lez Enfants, Thèse de Paris, No. 329, 1882.

A new interest was aroused by the masterly treatise of Still[15] with its report of cases of chronic joint disease of a special type. Still wrote of a chronic progressive enlargement of the joints and enlargement of the glands and spleen, the onset usually taking place before the period of second dentition. The tissue change seemed to be around the joint rather than in it. In Still's cases the hands were rarely involved. Muscle wasting took place early. It seemed impossible, according to him, to find a definite cause for the slight fever which usually existed. Adherent pericardium was noted in five cases. It is important to emphasize the fact that the author clearly understood that he was not dealing with rheumatoid arthritis which also occurred in childhood and which resembled the rheumatoid arthritis of adults, for the term "Still's" disease has been applied rather loosely to all cases of chronic arthritis and periarthritis of childhood.[16]

Reports of cases of deforming arthritis of the type described by Still commenced to appear after the publication of the original article. Koeppe[17] spoke of three cases which showed pericardial adhesions, and in the one case which came to necropsy no evidence of tuberculosis was found. Weber[18] believed that Still probably took too gloomy a view of the prognosis. Valvular disease was present in his cases, but no cultures were obtained from the joints. In one case, a positive reaction was obtained with Koch's tuberculin. In Edsall's[19] case a positive tuberculin reaction was obtained, and tubercle bacilli were found in the lymph nodes.

Poncet,[20] as a result of studies made since 1897, believed that chronic arthritis was the consequence of a general and attenuated infection, and that these larval infections might have their beginning in infancy or adolescence, and while they might be caused by syphilis, nevertheless, the most common cause must be tuberculosis. This conception of a "rheumatisme tuberculeux" has been the subject of lively controversy, but has not found general acceptance. Garrod[21] and

15. Still, G. F.: On a Form of Chronic Joint Disease in Children, Med. Chir. Tr., Edinburgh **80**:47, 1897.

16. In the Surgeon-General's Catalogue, all cases of arthritis deformans were classified under the heading "Still's Disease."

17. Koeppe, H.: Jahrb. f. Kinderh. u. Physische Erziehung, Vienna and Leipsic **76**:707, 1912.

18. Weber, F. P.: Still's Type of Chronic Joint Disease in Children and the So-Called Tuberculous Rheumatism, Brit. J. Child. Dis. **2**:208, 1905.

19. Edsall, D. L.: Concerning the Nature of Still's Type of Chronic Polyarthritis in Children, Arch. Pediat. **21**:175, 1904.

20. Poncet: Arthrites Chroniques et Rheumatisme Tuberculeux, Presse méd. **21**:244, 1913.

21. Garrod: Allbutt's System of Medicine, Ed. 2, **3**:17, 1908.

Ibrahim[22] express special doubt as to the justification of regarding "Poncet's disease" as a separate clinical entity.

The same difficulties, although possibly in less degree, have been encountered in attempts at classification. Rachford[23] mentions three types: the first, with hypertrophic changes predominant; the second, with atrophic changes prominent, and the third, Still's disease. One of the most complete clinical studies of the disease is that of Spitzy.[24] He reports cases and gives an excellent bibliography.

The question of the etiology of arthritis deformans in children has gone through the same stages of development as it has in adults. For a long time, and in spite of the fact that acute arthritis had been known to belong to a group of metastatic phenomena following tonsillar infection, chronic joint troubles were believed to be due to metabolic disturbances, although Still considered the possibility of infection as a cause of the type of disease which he observed. In addition to errors in metabolism, the nervous system and the ductless glands were also considered in attempting to assign a cause of the disease.

Royal Whitman,[25] in an interesting philosophical discussion of the disease, stated that "if rheumatoid arthritis is to be considered as of infectious nature, its distinction over ordinary forms of infection, whether local or general, is certainly marked. . . . One must presuppose a focus of infection within the body which is constantly active." Billings[26] and his co-workers were among the first to put this view into practical application. Rosenow[27] examined the lymph glands which drain the affected joints and found streptococci in them. He also found endothelial proliferation of the blood vessels which might have led to the trophic changes commonly seen. Lindsay[28] reported nineteen cases of rheumatoid arthritis in children and emphasized foci of infection as being etiologic factors. While he recommended that there should be a systematic examination of all the orifices of the body — mouth, nose, ears, rectum and vagina — and urged that the infected focus, if found, be eradicated, he did not say that by such eradication the patient was benefited.

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22. Ibrahim: Handb. d. Kinderh., Pfaundler and Schlossmann, Ed. 2 **2**: 401, 1910.

23. Rachford: Diseases of Children, 1912, p. 408.

24. Spitzy: Zur Chronischen Arthritis der Kinder, Ztschr. f. orthop. Chir. **11**:699, 1903.

25. Whitman, R.: A Report of Final Results of Two Cases of Polyarthritis in Children of the Type First Described by Still, Together with Remarks on Rheumatoid Arthritis, Med. Rec. **63**:601, 1903.

26. Billings, F.: Chronic Focal Infections and Other Etiological Relations to Arthritis and Nephritis, Arch. Int. Med. **9**:484 (April) 1912.

27. Rosenow, E. C.: Etiology of Arthritis Deformans, J. A. M. A. **62**:1146 (April 11) 1914.

28 Lindsay, J.: Rheumatoid Arthritis in Children, Edinburgh M. J. **10**:332, 1913.

Hammond's[29] experience with tonsillectomy in arthritis deformans did not lead him to feel encouraged as to the effectiveness of this operative measure. This is in accord with our own experience. A possible relationship between sinus infection and arthritis deformans in adults has been assumed by workers following the suggestions of Billings and his co-workers. The results of operation on the sinuses have not been uniformly satisfactory. It is interesting to note that chronic sinusitis was found in one instance in a series of cases of arthritis deformans in swine studied by Segikuchi and Irons.[30]

Experience in this clinic[31] early demonstrated that mere removal of the tonsils and adenoids was not sufficient to control the symptoms and signs of the disease, or to arrest its progress; therefore, it became necessary to institute a search for another focus of infection, especially as it was usual for such manifestations as slight elevations of temperature, leukocytosis, exacerbations of pain and even involvement of new joints to persist. No trouble was found in the teeth in the cases studied, and attention was then directed to the nose and especially to the accessory nasal sinuses, the details of the work being reported elsewhere by Dr. L. W. Dean,[32] whose investigations and operations have made this article possible. A brief summary of the cases studied since 1916 is appended herewith.

REPORT OF CASES

CASE 1.—D. M., aged 5 years, was admitted in September, 1916, having had "joint" trouble for two years previously. In this case the spleen was palpable. Elbows, hips, ankles, dorsal spine and to some extent the hands and fingers on both sides were involved. The tonsils and adenoids were removed two months after admission, but improvement was extremely slow and a few slight exacerbations were noted. Sinus operation was not done because at this time the relationship between nose infection and joint trouble was not realized. After three years there was a distinct recurrence of the trouble. The patient now shows distinct evidence of chronic infection which can only be situated in the nasal sinuses as present examination shows.

CASE 2.—J. B., aged 1½ years, had been ill four months before admission to the hospital in May, 1917. The onset of the disease was subacute, the arms, elbows and knees being involved. The spleen was not palpable, and no lymph glands were felt. The joints were distinctly tender, and there was painful

29. Hammond, R.: The Rôle of the Nose, Throat and Accessory Sinuses in the Etiology of Chronic Infectious Arthritis, J. A. M. A. **65**:1091 (Sept. 25) 1915.

30. Segikuchi and Lyons: Chronic Arthritis in Swine, J. Infect. Dis. **21**:526 (Dec.) 1917.

31. Byfield, A. H.: Systemic Manifestations of Chronic Nasal Sinus Infection in Childhood, J. A. M. A. **71**:511 (Aug. 17) 1918.

32. Dean, L. W.: Infections of the Paranasal Sinuses in Infants and Young Children with Special Reference to Adenoids and Chronic Tonsillitis as Etiologic Factors, Ann. Otol., Rhinol. & Laryngol. **27**:534 (June) 1918; Dean, L. W.: Nasal Sinus Disease in Infants and Young Children, Including Bacteriologic Study, Ann. Otol., Rhinol. & Laryngol. **28**:454 (Sept.) 1919.

limitation of motion. A leukocytosis of 21,800 was observed. The highest temperature noted was 99.8 F. Three weeks after admission an operation for removal of tonsils and adenoids was performed. Improvement was very marked and rapid and by July 11 (five weeks later) pain and tenderness had practically disappeared. Some ankylosis persisted. There were no recurrences.

CASE 3.—J. N., male, aged 11 years, had been sick fourteen months before coming to the hospital in July, 1917. The onset was subacute, and at the time of admission the spleen was not felt. There was marked pain on handling, marked deformity and limitation of motion of many joints. The test for tuberculosis was practically negative. Six months after admission, the tonsils and adenoids were removed, but the benefit was not great. Seven weeks later a nasal sinus operation was performed and a very striking diminution of pain was observed in less than one week. The anorexia so often present in these cases diminished and the patient's general health improved. A second operation on the nasal sinuses was found necessary three months following the first. Again, improvement was noted within a day. From that time until the present there has been no exacerbation, and the patient, while still having bony deformity, must be regarded as having had his trouble definitely arrested. An older brother, not treated, is in an unbelievably crippled condition.

CASE 4.—F. R., aged 7 years, had been ill three years before admission in September, 1917. The onset was with sore throat, with progressive involvement of the joints following this. On admission there was torticollis, the cervical lymph glands enlarged, the spleen was very slightly palpable but not reaching beyond the costal margin, and there was marked involvement of many joints with swelling, deformity, limitation of motion and distinct pain on handling. The tonsils were removed three weeks after admission. Following this operation only slight improvement was noted. The sinuses were operated on only a short time after the tonsil operation. In this case the temperature elevation persisted, rising as high as 103 F. with a leukocyte count of 25,000. When the child was seen again about six months later, the fever had disappeared and no acute or subacute condition was present in the joints. A final observation in 1919 showed that the disease was distinctly arrested with only a residue of joint involvement undergoing orthopedic treatment. In this case, the operation on the nasal sinuses did not give as quick results as were seen in other cases, but the benefit to the joints was very great. The final result must be regarded as eminently satisfactory in view of the fact that there was a complete cessation of the recurring attacks, together with a partial return of function. The fact that the temperature elevation persisted even after the sinus operation suggests that a complete removal of infected tissue was not effected.

CASE 5.—M. R., aged 9 years, was admitted to the hospital in October, 1917, after seven years of recurring joint attacks. The usual findings of joint deformity and tenderness were present. The tonsils and adenoids were removed and sinus operations were done on the same day, and while there was a marked improvement, it was impossible to clear up the nasal infection even six months after admission. The patient, however, was without joint trouble for nearly two years, when he was readmitted with a recurrence. It should be mentioned, however, that in addition to the arthritis deformans, there were present a nephritis and a recurring colitis as complications. Furthermore, the child reacted very strongly to the intradermal tuberculin test. We must regard this case as one in which the patient was benefited but not cured. The complicating infection may have been responsible for the unsatisfactory result.

CASE 6.—F. B., aged 12 years, was sick one year before admission to the hospital in July, 1917. This case is reported mainly to emphasize the fact that removal of the tonsils and adenoids is insufficient to control the disease. This patient had only one or two of the larger joints involved and because of improvement in his general condition following the removal of the tonsils and adenoids, with arrest of the arthritic process, he was discharged, as it was

believed that the respiratory infection had worn itself out. The incorrectness of our assumption was shown by the fact that the patient returned to the hospital seven months afterward with many joints involved and with much resultant crippling. Operation on the sinuses was performed but the boy was removed from the hospital before it was possible to determine the permanent benefit obtained, although the immediate improvement was striking. In the light of our experience during the past three years, it seems fair to believe that had more radical treatment of the nose been instituted at once, spread of the disease could have been prevented.

CASE 7.—W. F., aged 11 years, had been sick three years before coming here in July, 1918. The onset was insidious. Few joints were involved and the existing deformity was slight. An incomplete tonsillectomy had been performed two years before admission and it was necessary to remove the tonsil stumps. This last operation was followed with infection of some joints previously free. Pain on handling and limitation of movement were quite constantly present during the three months that the patient was under observation. Frequent examinations were made. Operation on the nose was then done, following a submucous resection and this was followed in a week by unmistakable local and general improvement; from this period until the present there has been no recurrence and the patient has needed only orthopedic correction of the deformity already present.

CASE 8.—E. T., aged 3 years, was sick four months before admission in November, 1918, beginning with tonsillitis and marked multiple joint involvement, with very distinct tenderness and deformity. Tonsillectomy was performed shortly after admission, and the patient has had no recurrence after one year. The improvement was rapid and continuous.

CASE 9.—H. H., aged 8 years, when admitted in March, 1919, had been sick one year previously. The onset in this case was rather sudden, following tonsillitis. Few joints were involved, but there was trouble in the wrist and in some of the small bones of the left hand. A tonsil operation had been done; but, one year after this it was necessary to have the tonsil stumps removed and a nasal sinus operation was performed at the same time. There was moderate improvement for four months when there was a recurrence and a torticollis developed in addition to trouble in the hand and in the foot. The girl was put in bed and observed for nearly a month. A cast was applied to the foot, but the condition remained unchanged. While the distress and pain were not great, it was continuous and distinctly affected the girl's general health. There was limitation of motion in the neck and pressure on the hands was painful enough to cause crying. A second and more complete nasal operation was then done, and within a few weeks the torticollis had entirely disappeared, movement of the hands was possible, with practically no pain and the foot was much improved. The effect of this operation on the general health of the patient was a surprising one, even to those who have had occasion to share in the observations made in the past.

CASE 10.—I. H., aged 3 years, was sick one year before admission to the hospital in March, 1919. The tonsils and adenoids were removed and this was followed with rapid improvement and no recurrence.

DISCUSSION

In these patients a uniformity in the constitutional signs and symptoms was manifest, there being present emaciation of varying degree, anorexia and irregularity of temperature with frequent rises to 100 F. and occasional rises to 103 F. A leukocytosis of from 11,000 to 25,000 was present. At the time of admission to the hospital the spleen was

practically never found to be enlarged. A positive tuberculin test was seen in one case only. A rheumatic history was obtained in a few cases, but in only one instance (Case 3) was more than one member of the family similarly affected. The teeth were examined in every case and only once were they found to be diseased. No benefit followed extraction. Blood cultures were taken in two cases and were negative.

The disease tended to have its onset in the larger joints, usually the knee or the ankle, while the bones and articulations of the wrist were affected later. In two patients the affection remained monarticular for so long a period as to suggest a diagnosis of tuberculosis of the joints. With the progress of the disease to other joints the true nature of the trouble was indicated and appropriate treatment instituted.

The cases under discussion, therefore, do not appear to belong to the group described by Still, but resemble more nearly those cases which are commonly referred to as rheumatoid arthritis. In one case (Case 5) a very positive Mantoux reaction was obtained so that a diagnosis of Poncet's disease might have been considered. Since, however, two operations on the sinuses were followed with a subsidence of joint swelling and pain, it seems more probable that we were dealing with a case of deforming arthritis in an individual affected with tuberculosis. Convalescence in this case was slower than in most of the others; it may be possible that the presence of the tuberculous infection tended to delay improvement. In view of the fact that there has been considerable discussion as to the nature of Poncet's disease, this case is worthy of note.

There is no great choice of therapeutic measures in arthritis deformans in children if the infectious nature of the disease is admitted and if it is granted that the accessory sinuses of the nose are the commonest seat of the etiologic focus. General supportive measures, with orthopedic treatment of the joints alone, produce too little and too slow improvement to be regarded as efficacious. Furthermore, an extension of the disease cannot be thus avoided. Removal of the tonsils and adenoids is followed by slow improvement, but does not guarantee that there will not be a relapse. Intravenous and intramuscular injection of foreign proteins has not been made here, except in two cases (Cases 3 and 6) and in neither of these was the improvement striking. That one injection was not sufficient to prevent a very marked extension of the disease was shown in Case 6. It should be stated, however, that without a more careful trial, a definite opinion cannot be given. In the children in this series less than 3 years of age, removal of the tonsils and adenoids alone has seemed to be sufficient to cure and arrest the progress of the disease. It appears, therefore, that the younger patients with this malady may be spared nasal intervention. In older children, however, removal of the tonsils and

adenoids is definitely insufficient to accomplish this end, and in them the surgical intervention on the nasal sinuses, as practiced by the nose and throat department, is indicated and has yielded results which are eminently satisfactory.

It will be admitted that in estimating the benefits following on operative treatment of the nose, there is a considerable personal element: improvement, like beauty, may be in the eye of the observer. Past experience, however, with cases of arthritis deformans which were treated expectantly, with plaster casts, or by tonsillectomy, made tolerably clear the fact that where improvement did take place, it was slow and slight. It was this observation which led to the request that a search be made for other possible foci of infection, since it was believed that the older methods of treatment were unsatisfactory.

In spite of the fact that the disease has been termed a periarthritis from a pathologic standpoint, there is little question that the joint tissues adjacent to the cartilage show an extensive involvement,[33] and it can scarcely be expected, therefore, that a complete restitution to normal is possible, especially when the disease has existed for a number of years. Furthermore, the difficulty of complete elimination of foci of infection is also undeniable. When, however, nasal operations were almost invariably followed within a week by unmistakable subsidence of symptoms and signs, when "flare ups" occurred, if at all, only at very long intervals, when a very rapid improvement was noted in the general health of the child as well, it could scarcely be denied that there must be some direct connection between the operation and the improvement achieved. The results were immeasurably better than were those following the usual methods of treatment.

It should be borne in mind, also, that orthopedic treatment is definitely indicated where marked deformity and marked joint pathology exists, but the extent to which restitution may take place when the infection is controlled, is surprising even if casts are not applied. Five patients have been free from recurrence for at least two years.

CONCLUSIONS

From a study of the cases reported it may be concluded that:

1. Arthritis deformans in children results chiefly from a chronic infection situated in the tonsils and adenoids and in the accessory sinuses of the nose.

2. In children less than 3 years of age the portal of infection seems to be limited to the tonsils and adenoids. After this time removal of tonsils and adenoids is ineffective in arresting the progress of the disease.

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33. Axhausen: Die Deformierende Gelenkentzündung (Arthritis Deformans) Im Lichte Neuer Forschung, Berl. klin. Wchnschr. **52**:1205, 1915.

3. A sinus infection should be suspected as an etiologic factor if, after the tonsils and adenoids are removed, there remains elevation of temperature (even if slight), leukocytosis, poor appetite, together with a slowness of the joints to become less painful and swollen. Relapse and exacerbations are definite indications of the need of nasal treatment.

4. Poncet's disease is probably no more than arthritis deformans in an individual infected with tuberculosis.

5. Although supportive and orthopedic measures are helpful, surgical treatment of the nasal sinuses is to be regarded as the most important therapeutic measure indicated in arthritis deformans in children.

6. The prognosis in uncomplicated cases is good as far as arrest of the disease is concerned. The deformity and functional disability may persist for a considerable time.

CALCIUM METABOLISM OF INFANTS AND YOUNG CHILDREN, AND THE RELATION OF CALCIUM TO FAT EXCRETION IN THE STOOLS*

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PART I. INFANTS TAKING MODIFICATIONS OF COW'S MILK

Recently a large number of stools from children who were receiving food of a varied character were collected for a study of fat metabolism of infants and young children. In nearly all of this material total ash and calcium determinations were made. It was believed that the large number of observations made would furnish data of value in answering some much debated questions regarding calcium metabolism, especially in its relation to fat metabolism.

Many investigators have published data and have advanced theories on this subject, but the conclusions reached are contradictory and in many cases the observations on which they are based are very few in number. For example, the observations made on one breast fed infant for a single period and on one child fed on cow's milk, also for a single period, are quoted in nearly every article on this subject, and have been taken as standards for normal calcium metabolism.

Many diverse views are prevalent in regard to calcium metabolism. Some writers believe that a liberal intake of calcium results in an excessive excretion of calcium which is accompanied by a harmful loss of fat. Others hold that a high fat intake induces a large fat excretion, accompanied by a serious loss of bases, especially calcium. Both these dangers are supposed to be associated with occurrence of soapy stools. The German literature especially has emphasized the pathologic significance of large light-colored crumbly stools rich in calcium soaps and calcium phosphate, a type of stool apparently not so frequently seen in this country. There are still other writers who think that a large intake of calcium leads to a storage of calcium in the body which may have injurious effects. On the other hand, there are many who believe that an ample intake of both fat and calcium produces only beneficial results. It is generally accepted that cod liver oil has a

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beneficial effect on the retention of calcium, but this opinion seems to be not entirely unanimous. The literature on all these questions is so voluminous that there is not space here to discuss it in detail.

The points of general interest in regard to calcium metabolism may be stated in the form of the following questions.

1. What is the normal excretion and absorption of calcium by children taking (a) modifications of cow's milk, (b) mixed diet?

2. How is the calcium excretion and absorption affected by the amount of (a) the calcium intake, (b) the fat intake?

3. Is there any evidence that either an excessive calcium intake or a very small calcium intake is harmful?

4. Is there any relation between the age or the weight of the child and the amount of calcium absorbed normally?

5. Is there any constant relation between the excretion of calcium and the excretion of fat in the form of soap?

6. Is there a serious loss of calcium in soapy stools?

7. On what is the calcium percentage of the total solids of the stool dependent?

8. How do the calcium excretion and absorption differ from normal when children are suffering from (a) diarrhea, (b) chronic intestinal indigestion, (c) active rickets or (d) when recovering from rickets?

9. What is the effect on calcium metabolism of (a) cod liver oil, (b) of vegetable fats?

In the observations reported in this and a subsequent paper on this subject the period of stool collection and the amount of food intake were exactly known. Thus, it was possible to determine closely the amount of calcium excreted and absorbed daily. In most of these cases values for the urinary excretion of calcium were not obtained. Hence, the actual *retention* of calcium is not reported. However, the *absorption* is practically the same as the retention, since the excretion of calcium in the urine of infants and young children is normally very small in amount. Previous work done in this laboratory has shown a general range of from 0.01 to 0.05 gm. of calcium oxid (CaO) excreted daily in the urine of infants taking modifications of cow's milk and from 0.03 to 0.15 gm. for children taking a mixed diet. With the small number of breast fed infants for whom values have been obtained, the urinary excretion of calcium was found to be from 0.014 to 0.026 gm. of calcium oxid daily. Rachitic children were found to excrete even less calcium oxid in the urine than did normal children of the same age. By calcium *excretion* is meant the total amount of calcium lost in the stools. Whether part of this calcium has been absorbed and subsequently excreted into the large intestine, as is believed by many to be the case, is not taken into consideration.

TABLE 1.—Normal Infants. Relation of Absorption of Calcium to Calcium and Fat Intake

No.	Case	Age in Mos.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Per Cent. of CaO Intake Absorbed	Intake of Fat Gm. per Kg.	Grams of CaO Intake per Gm. Fat Intake	Remarks
113	J. S. 1	7.5	4,060	Nonhomogeneous	0.488	0.099	20.2	6.6	0.074	Skimmed milk with large amount of olive oil
72	A. K.	5.5	3,910	Constipated	0.297	0.133	44.8	5.6	0.053	
58	J. I.	12	5,485	Normal	0.285	0.131	46.1	5.2	0.055	
65	P. D. 3	7.5	4,884	Constipated	0.285	0.068	23.8	4.9	0.058	Rather high carbohydrate in diet
54	R. C.	8.5	6,290	Normal	0.283	0.124	43.8	5.2	0.054	High calories per kilogram
86	J. M.	4	3,662	Softnormal	0.279	0.098	35.3	3.7	0.076	Dried milk formula
158	H. B.	13	6,935	Normal	0.266	0.043	16.4	5.0	0.053	Very large stools
57	M. S. 2	8	6,145	Normal	0.266	0.123	46.6	4.8	0.055	
70	P. D. 1	7	4,635	Constipated	0.264	0.095	38.5	4.9	0.053	Rather high carbohydrate in diet. High calories per kilogram
71	P. D. 2	7	4,679	Constipated	0.257	0.030	11.7	4.8	0.053	Rather high carbohydrate in diet. High calories per kilogram
82	P. L.	4	5,055	Nonhomogeneous	0.235	0.099	42.0	3.1	0.076	Dried milk formula
97	B. W. 1	15	5,873	Softnormal	0.231	0.117	50.8	4.7	0.049	Recovering from multiple fractures. Very low calories per kilogram
85	C. M.	2	3,450	Nonhomogeneous	0.229	0.116	50.7	4.2	0.055	
166	A. A.	7	3,489	Nonhomogeneous	0.224	0.063	28.2	3.3	0.068	Very high proportion carbohydrate in diet
63	F. N. 2	11	5,800	Softnormal	0.222	0.093	41.9	4.2	0.054	High carbohydrate in diet. High calories per kilogram
60	F. N. 1	11	5,620	Constipated	0.217	0.090	45.1	4.7	0.047	High carbohydrate in diet. High calories per kilogram
62	J. M.	7	5,900	Normal	0.204	0.063	30.8	4.2	0.049	
126	W. H. 4	14	6,467	Nonhomogeneous	0.195	0.130	66.7	5.5	0.035	Recovering from rickets. Taking cod liver oil
49	G. H.	7.5	5,660	Constipated	0.193	0.090	46.8	9.5	0.023	Top milk formula. Cod liver oil. Very high calories per kilogram
119	W. H. 5	14	6,536	Nonhomogeneous	0.191	0.115	60.5	3.9	0.049	Recovering from rickets. Cod liver oil just discontinued
101	J. K. 2	16	4,720	Softnormal	0.180	0.083	45.9	3.5	0.052	Underfed
67	M. R.	6	6,860	Constipated	0.179	0.042	23.6	3.4	0.053	
81	R. B.	15	9,075	Normal	0.177	0.097	55.0	3.4	0.052	15 c.c. milk of magnesia
66	P. S. 3	10	6,560	Softnormal	0.167	0.064	38.2	3.6	0.046	High proportion carbohydrate in diet
96	R. L. 1	12	10,400	Normal	0.152	0.069	45.3	2.9	0.053	Very low calories per kilogram
92	L. B.	3.5	3,535	Nonhomogeneous	0.148	0.051	34.6	3.5	0.042	Sweetened condensed milk. Very high proportion carbohydrate
83	A. P.	6	6,540	Softnormal	0.136	0.070	51.7	2.3	0.059	Very low calories per kilogram
50	B. P.	10	9,574	Normal	0.088	0.014	15.2	3.8	0.023	Top milk formula. Low calories per kilogram
68	M. M.	11	11,490	Normal	0.087	0.013	16.0	2.1	0.043	Much underfed. Rather high proportion carbohydrate in diet
94	E. W.	5	3,770	Softnormal	0.069	—0.064	0.0	0.9	0.077	Malted milk. Extremely high proportion carbohydrate in diet

By calcium *absorption* is here meant the difference between the calcium intake and the excretion of calcium in the stools.

The values for ash and calcium in the feces were obtained by analysis of the dried material. The ash was determined by the Stölte method,[1] which consists of the application of heat to the finely ground material in a platinum dish inside a porcelain or silica dish, without the addition of acid or alkali. The calcium was determined in the weighed ash by the McCrudden method.[2]

In some of the cases, especially when the children were taking a mixed diet, the calcium in the food was not determined by direct analysis, but was estimated from the known weight or measurement of the various articles of the food intake. The calcium content of some of the articles of food which made up the diet had previously been determined in this laboratory.

In this article are presented only the findings for infants taking modifications of cow's milk. The data for children taking mixed diet will be discussed in a later paper.

HEALTHY INFANTS

The first group of infants here considered were normal or approximately normal as to their digestion. Most of these children were healthy and gaining weight at the time of observation. None of them had diarrheal stools. Table 1 shows the relation of the calcium absorption to the calcium and the fat intake for the group. These values are expressed as grams per kilogram of body weight. Only in this way could the intake for infants of widely differing weights be correlated. A study of the values for total intake and absorption of calcium, weight not being considered, showed no consistent variation in absorption as related to intake.

Calcium Absorption and Its Relation to Calcium Intake. — The intake of calcium oxid per kilogram ranged from 0.13 to 0.30 gm., with an average of 0.22 gm. in twenty-six of the thirty cases studied. Of the remaining four cases, in one the intake of calcium oxid was extremely high, and in the others it was very low. In twenty instances the intake of calcium oxid was more than 0.19 gm.

The relation of the calcium absorption to the calcium intake is summarized in Table 2. It is here seen that the best absorption occurred when the intake exceeded 0.19 gm. per kilogram, and that when the intake was less than 0.10 gm. the absorption was very poor.

1. Stölte: Biochem. Ztschr. **35**:104, 1911.
2. McCrudden: J. Biol Chem. **10**:187, 1911.

In the one case with exceptionally high intake, the absorption was only 0.099 gm. per kilogram, which was lower than in many cases where the intake was less.

The average absorption of calcium oxid in all the cases in which the intake exceeded 0.10 gm. per kilogram, was 0.089 gm. per kilogram. This value is higher than that found for breast fed infants. With five breast fed infants, having an intake of calcium oxid ranging from 0.045 to 0.097 gm. per kilogram and averaging 0.081 gm., we found an absorption of from 0.035 to 0.071 gm. per kilogram averaging 0.054 gm. In the much quoted case reported by Blauberg[3] the intake of calcium oxid was 0.041 gm. per kilogram and the absorption was 0.031 gm. per kilogram.

TABLE 2.—RELATION OF CALCIUM ABSORPTION TO CALCIUM INTAKE

CaO Intake per Kg.	No. of Cases	Absorption of CaO Over 0.09 Gm. per Kg.	Absorption of CaO, 0.06 to 0.09 Gm. per Kg.	Absorption of CaO Under 0.06 Gm. per Kg.
Over 0.19 gm.	20	15	3	2
Under 0.19 gm.	10	1	4	5
Under 0.10 gm.	3	0	0	3

Relation of Calcium Absorption to Fat Intake.—In twelve of the sixteen cases in Table 1 in which the absorption of calcium oxid exceeded 0.09 gm. per kilogram, the intake of fat exceeded 4.2 gm. per kilogram. In the other four cases in which the absorption of calcium oxid exceeded 0.09 gm. per kilogram the fat intake was less than 4.2 gm. per kilogram, but other factors were present which may have influenced the calcium absorption. One child had been receiving cod liver oil up to the time of the observation; another child had received one-half ounce of milk of magnesia; the other two children were taking a dried milk formula, in which the calcium may be present in a more easily assimilated form as a result of the treatment to which the milk has been subjected during the process of drying. In only four of the fourteen cases in which the absorption of calcium oxid was less than 0.09 gm. per kilogram was the fat intake as great as 4.2 gm. per kilogram.

In two-thirds of the cases in the table the food contained from 0.045 to 0.060 gm. of calcium oxid for every gram of fat. When the calcium oxid per gram of fat in the intake was less than 0.045 gm. per kilogram, the only high absorption of calcium oxid occurred when cod liver oil was being taken. This optimum ratio of fat and calcium intake existed in some cases when the intake of both calcium and fat was too low. It has been noted that the best calcium absorption took

3. Blauberg: Ztschr. f. Biol. **40**:36, 1900.

place when the intake of fat was 4.2 gm. or more per kilogram. The lowest value for calcium oxid intake per kilogram which would maintain the optimum ratio of calcium oxid to this amount of fat in the intake therefore would be 0.19 gm. per kilogram, a value already noted as the amount necessary to secure good absorption.

Percentage Absorption of Calcium.—From 35 to 55 per cent. of the calcium intake was absorbed in eighteen of the thirty cases. The only higher values for percentage absorption were noted twice with a child who was recovering from rickets and who had been receiving cod liver oil for a long period. A higher percentage of the calcium intake is absorbed by breast fed infants. In our cases the average for breast fed infants was 66.7 per cent.; in Blauberg's case the absorption was 75.6 per cent.

Calcium Requirement.—The average absorption of calcium oxid found for five breast fed infants was 0.054 gm. per kilogram. Since, according to our observations, infants taking modifications of cow's milk absorbed on the average only about 45 per cent. of the calcium intake, it is necessary to provide for them a minimum intake of about 0.130 gm. of calcium oxid per kilogram to insure even the low average absorption of breast fed infants; there would, therefore, seem to be danger in an intake lower than this. On the other hand, there appears to be no advantage in an intake greater than 0.30 gm. per kilogram. Hoobler[4] says that anything less than 1.0 or 1.5 gm. of calcium oxid daily should be considered a calcium poor intake for infants taking modifications of cow's milk. His requirement, calculated for an infant of seven kilograms body weight, would be from 0.143 to 0.215 gm. of calcium oxid per kilogram of body weight. This range includes our above noted value, 0.19 gm., the intake of calcium oxid per kilogram found to be necessary to secure good absorption.

Other Factors in Relation to Calcium Absorption.—The values presented in Table 1 show that there was no definite variation in calcium absorption per kilogram according to the age or weight of the child.

There was also no definite relation between the type of stool and the amount of calcium oxid absorbed per kilogram or the percentage of the calcium intake absorbed.

Table 3 shows for the cases considered in Table 1 the calcium excretion in the stools in relation to the actual calcium and the fat intake; also the relation of the calcium excretion to the excretion of fat and of soap in the stools.

Calcium Excretion and Its Relation to Calcium Intake and to Fat Intake.—With the exception of two unusually high values the excre-

4. Hoobler: Am. J. Dis. Child. **2**:107, 1911.

tion of calcium in the stools ranged from 0.34 to 1.06 gm., with an average of 0.70 gm. As a rule, the higher the calcium intake the higher the calcium excretion; the lower the intake, the lower the excretion. In general, the fat intake and the calcium intake were parallel, so that a high calcium excretion occurring with a high fat intake could be accounted for by a high calcium intake. When a high fat intake accompanied a low calcium intake, the excretion of calcium was low, except in one case. Hence, there is little evidence that there is any constant relation between the amount of the fat intake and the amount of calcium excreted in the stools.

TABLE 3.—CALCIUM EXCRETION OF HEALTHY INFANTS IN RELATION TO ACTUAL CALCIUM AND FAT INTAKE

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap in Stools, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
113	J. S. 1	Nonhomogeneous	1.98	26.6	1.58	1.53	0.26	1.6	1.3
158	H. B.	Normal	1.84	34.7	1.54	4.93	3.90	25.3	21.2
71	P. D. 2	Constipated	1.20	22.6	1.06	3.34	2.64	24.9	22.0
65	P. D. 3	Constipated	1.39	24.0	1.06	2.95	2.05	19.3	14.7
54	R. C.	Normal	1.78	32.8	1.00	2.70	1.97	19.7	11.1
67	M. R.	Constipated	1.23	23.4	0.94	2.79	2.25	24.0	18.3
62	J. M.	Normal	1.20	24.4	0.89	1.14	1.01	11.4	9.6
57	M. S. 2	Normal	1.63	29.4	0.87	3.36	2.42	27.8	14.9
96	R. L. 1	Normal	1.58	30.0	0.86	2.04	1.47	17.0	9.3
58	J. I.	Normal	1.56	28.6	0.84	1.30	1.11	13.2	7.1
68	M. M.	Normal	1.00	23.3	0.84	1.24	0.96	11.5	9.6
70	P. D. 1	Constipated	1.22	22.9	0.78	2.21	1.67	22.3	13.7
63	F. N. 2	Softnormal	1.29	24.1	0.75	5.09	3.68	49.1	28.6
81	R. B.	Normal	1.60	31.0	0.72	3.91	3.48	48.3	21.7
50	B. P.	Normal	0.84	36.5	0.71	2.91	2.01	29.2	24.7
82	P. L.	Nonhomogeneous	1.19	15.7	0.69	1.73	1.12*	16.3*	9.4*
66	P. S. 3	Softnormal	1.10	23.7	0.68	0.85	0.30	4.4	2.7
97	B. W. 1	Softnormal	1.36	27.6	0.67	2.54	1.81	27.6	13.3
60	F. N. 1	Constipated	1.22	26.2	0.67	4.12	3.31	49.4	27.1
86	J. M.	Softnormal	1.02	13.4	0.66	2.08	1.67	25.3	16.4
72	A. K.	Constipated	1.16	22.1	0.64	3.04	2.22	34.7	19.1
49	G. H.	Constipated	1.09	48.3	0.58	2.98	2.33	40.2	21.4
166	A. A.	Nonhomogeneous	0.78	11.4	0.56	2.46	0.04	0.7	0.4
94	E. W.	Softnormal	0.26	3.4	0.50	1.00	0.75	15.0	29.0
119	W. H. 5	Nonhomogeneous	1.24	25.2	0.49	2.64	0.78*	15.9*	6.3*
101	J. K. 2	Softnormal	0.85	16.5	0.46	0.66	0.46	10.0	5.4
83	A. P.	Softnormal	0.89	15.1	0.43	0.65	0.50	11.6	5.6
126	W. H. 4	Nonhomogeneous	1.26	35.8	0.42	3.35	1.69*	40.2*	13.4*
85	C. M.	Nonhomogeneous	0.79	14.4	0.39	1.64	1.05*	26.9*	13.3*
92	L. B.	Nonhomogeneous	0.52	12.5	0.34	2.06	1.38*	40.7*	26.6*

* Value possibly too high. Stools acid.

Relation of Calcium Excretion to Excretion of Total Fat and of Fat as Soap.—There was no constant relation between the excretion of total fat and the excretion of calcium in the stools. In order to study the relation between the excretion of calcium and that of fat as soap, the findings shown in Table 2 were averaged by groups according to the type of stool, since the average soap excretion varies definitely with the type of stool. These averages (Table 4) show that the cal-

cium excretion was more closely related to the calcium intake than it was to the excretion of soap. In the constipated stools there was a greater soap content than in the normal stools, but a smaller content of calcium, corresponding to a smaller calcium intake. The group of softnormal stools had a much greater soap content than that of the nonhomogeneous stools, but here, again, a somewhat lower calcium content corresponded to a somewhat lower calcium intake.

TABLE 4.—Relation of Average Calcium Excretion to the Average Soap Excretion

Stools	No. of Cases	CaO Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat as Soap in Stools, Gm. Daily	CaO Possibly Held as Soap	CaO not Held as Soap	CaO Possibly Held as Soap, Percentage of	
							CaO in Stools	CaO Intake
Constipated	7	1.22	0.82	2.35	0.24	0.58	30.9	19.5
Normal	9	1.45	0.92	2.04	0.20	0.72	22.6	14.4
Softnormal	7	0.97	0.59	1.31	0.13	0.46	20.4	14.4
Nonhomogeneous	7	1.11	0.64	0.90	0.09	0.59	20.3	10.1

There is a prevalent opinion that in soapy stools an excessive loss of calcium occurs, which may take place either as calcium bound to the fat in the form of insoluble soaps or as calcium phosphate. To show to what extent calcium was lost as insoluble soaps, values have been included in Table 3 giving the percentage of the total calcium *excretion* which could possibly be held in the form of soap, and also the percentage of the calcium *intake* which could be lost in this way. The amount of calcium that could be bound as soap is approximately one-tenth the amount of soap in the stool, since the combining ratio of calcium to the higher fatty acids is about one to ten. (Combining weight of calcium oxid is 28; stearic acid, 284.) The calcium bound as soap was never found to be as much as five-tenths and seldom more than three-tenths of the total calcium excretion. In all the cases in which the calcium lost as soap was more than three-tenths of the total calcium excretion the *total* loss of calcium was not excessive. In no case was the calcium lost as soap equal to three-tenths of the calcium *intake*.

The average values according to type of stool, included in Table 4, show that when the stools were constipated a greater proportion of both the calcium excretion and the calcium intake was lost as soap, but that the total loss of calcium and also the calcium intake averaged distinctly less for this group than for the group of normal stools.

Table 4 also includes average values to show to what extent calcium could be lost as phosphate in the different types of stools. These values were obtained by subtracting from the average total calcium excretion the amount of calcium which could be held as soap, that is,

one-tenth of the value for soap excretion. The remaining amount of calcium in the stools is the maximum that could be held as phosphate. Thus, it is shown that the amount of calcium that could be held as phosphate was greatest in the group of normal stools, which had lower soap content than did the constipated stools. In fact, the average value for the calcium not held as soap for the group of constipated stools with the highest soap content was practically the same as that for the group of nonhomogeneous stools with the lowest soap content. These observations seem to establish quite definitely that in soapy stools there is not an increased excretion of calcium phosphate. The excretion of calcium not held as soap was influenced more by the amount of calcium intake than by any other factor.

RACHITIC INFANTS

The second group of infants considered were suffering from rickets. Several of them also had tetany, either mild or severe. None of these children had diarrheal stools. Table 5 shows the absorption of calcium per kilogram and its relation to the calcium and the fat intake.

Calcium Absorption.—The intake of calcium oxid per kilogram of body weight was, in general, lower and the range was narrower than was the case with normal infants. In the majority of the cases the intake was less than 0.19 gm. per kilogram, which we have noted as the minimum to insure good absorption by healthy infants. The absorption of calcium oxid per kilogram by the rachitic infants as a group was extremely poor. In only one observation was there good absorption, that is, more than 0.09 gm., the average found for normal infants. In only six of the twenty-three observations was the absorption as much as 0.06 gm. per kilogram. In the cases where there was fair absorption of calcium oxid, the children either had been taking cod liver oil or were at the time suffering from severe tetany. In the one instance in which the absorption was high the child had been receiving cod liver oil for two and one-half months. The two next highest values were found with a child who had been taking cod liver oil for about one week. In these three instances the fat intake per kilogram was the highest shown in the table and was accompanied by a high intake of calcium oxid. Although several children showing a very low absorption were at the time of observation taking cod liver oil, none had received it for more than a few days. Of five observations on children suffering from tetany with convulsions, in only one instance was the absorption of calcium oxid less than 0.05 gm. per kilogram, which is a value higher than the average for rachitic infants.

The average absorption for the group of rachitic infants was 0.042 gm. per kilogram, which is less than one half that found for the normal infants.

TABLE 5.—Absorption of Calcium by Rachitic Infants

No.	Case	Age in Mos.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Per Cent. of CaO Intake Absorbed	Intake of Fat. Gm. per Kg.	Grams of CaO Intake per Gm. Fat Intake	Remarks
390	J. D. 4	5	3,901	Softnormal	0.249	0.077	30.9	6.7	0.037	Mild tetany. Cod liver oil for about one week
401	E. C. 1	11	6,724	Nonhomogeneous	0.247	—0.013	0.0	5.0	0.049	
64	L. R. 2	12	3,883	Softnormal	0.242	0.136	56.2	6.3	0.038	Cod liver oil
402	L. R. 1	9.5	3,454	Loose	0.240	0.055	22.9	4.6	0.053	
388	J. D. 1	5	3,845	Loose	0.235	0.065	27.6	3.9	0.060	Tetany. One very severe glottis spasm
389	J. D. 3	5	3,976	Loose	0.228	0.084	36.8	5.1	0.045	Cod liver oil for few days before observation
397	L. E.	8.5	7,395	Nonhomogeneous	0.210	0.029	13.8	4.7	0.045	Mild tetany, marked rickets.
61	E. C. 2	14	7,789	Constipated	0.193	0.032	16.6	3.3	0.059	Rather high proportion carbohydrate in diet
400	H. S.	9	7,825	Nonhomogeneous	0.184	0.049	26.6	2.5	0.073	Protein milk. Recently changed from breast. Severe tetany
394	J. T. 1	9	6,734	Constipated	0.183	0.012	6.6	3.7	0.049	
74	E. C. 3	15	7,788	Softnormal	0.181	—0.008	0.0	2.6	0.067	Rather low calories per kilogram
395	J. T. 2	9	6,827	Constipated	0.177	0.036	20.3	3.7	0.048	Injection saline first day.
398	B. M. 1	7	5,646	Nonhomogeneous	0.165	0.063	38.1	3.8	0.044	Mild rickets, tetany
391	P. L. 1	10	9,612	Constipated	0.158	0.055	34.7	3.3	0.048	Recently changed from breast milk. High carbohydrate. Mild rickets
393	P. L. 3	10	9,560	Normal	0.153	0.019	12.4	3.2	0.049	High carbohydrate. Mild rickets
387	D. W. 3	5	5,733	Nonhomogeneous	0.153	0.038	24.8	4.0	0.038	Cod liver oil. Magnesium sulphate injection. Convulsions
392	P. L. 2	10	9,574	Constipated	0.152	0.057	37.5	3.2	0.048	Mild rickets. Injection of magnesium sulphate
386	D. W. 1	5	5,815	Nonhomogeneous	0.148	0.055	37.2	3.1	0.048	Frequent and severe convulsions. Recently changed from breast milk
155	D. W. 2	5	5,816	Nonhomogeneous	0.145	0.065	44.8	3.0	0.049	Low calories per kilogram. Tetany with convulsions
154	W. H. 1	12	7,048	Nonhomogeneous	0.133	—0.017	0.0	2.5	0.054	Rather low calories per kilogram
399	T. W.	9	5,144	Nonhomogeneous	0.124	0.020	16.1	2.4	0.051	Mild tetany
117	W. H. 2	12	7,140	Normal	0.117	0.020	17.1	3.6	0.033	Cod liver oil for two days before observation. Low calories per kilogram
396	G. S.	9.5	7,445	Nonhomogeneous	0.073	0.033	45.2	3.0	0.024	Recently changed from breast milk. Cod liver oil. Marked rickets. Mild tetany

The poor absorption of calcium in rickets is strikingly shown by the values for percentage absorption. In fifteen of the twenty-three cases the percentage absorption was lower than the usual range for normal children.

The intake of fat per kilogram as well as that of calcium per kilogram was, on the whole, lower with the rachitic than with the normal children. The relation of the calcium intake to the fat intake was in most cases similar to that of the normal group.

TABLE 6.—Calcium Excretion by Rachitic Infants

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap in Stools, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
401	E. C. 1	Nonhomogeneous	1.66	33.9	1.75	..	..	..	..
74	E. C. 3	Softnormal	1.41	21.1	1.47	2.71	1.76	12.0	12.5
397	L. E.	Nonhomogeneous	1.56	34.9	1.34	..	..	..	..
393	P. L. 3	Normal	1.40	30.1	1.28	..	..	..	..
61	E. C. 2	Constipated	1.50	25.5	1.25	1.71	1.12	9.0	7.5
394	J. T. 1	Constipated	1.23	27.1	1.16	..	..	..	..
154	W. H. 1	Nonhomogeneous	0.94	17.4	1.06	0.91	0.49	4.7	5.2
391	P. L. 1	Constipated	1.52	31.4	0.99	..	..	..	..
395	J. T. 2	Constipated	1.21	25.0	0.97	..	..	..	..
400	H. S.	Nonhomogeneous	1.44	19.7	0.93	..	..	..	..
392	P. L. 2	Constipated	1.46	30.2	0.91	..	..	..	..
117	W. H. 2	Normal	0.84	25.4	0.69	2.24	..	..	..
390	J. D. 4	Softnormal	0.97	26.2	0.67	..	..	..	..
388	J. D. 1	Loose	0.90	15.1	0.66	..	..	..	..
387	D. W. 3	Nonhomogeneous	0.88	23.0	0.66	..	..	..	..
402	L. R. 1	Loose	0.83	15.7	0.64	..	..	..	..
389	J. D. 3	Loose	0.90	20.1	0.57	..	..	..	..
398	B. M. 1	Nonhomogeneous	0.93	21.1	0.57	..	..	..	..
386	D. W. 1	Nonhomogeneous	0.86	17.9	0.54	..	..	..	..
399	T. W.	Nonhomogeneous	0.64	12.6	0.53	..	..	..	..
155	D. W. 2	Nonhomogeneous	0.84	17.2	0.46	3.73	2.06*	44.7*	24.5*
64	L. R. 2	Softnormal	0.94	24.6	0.41	3.00	1.21*	29.5*	12.9*
399	G. S.	Nonhomogeneous	0.55	22.6	0.30	..	..	..	..

* Value possibly too high. Stools acid.

Table 6 gives for the rachitic children the calcium excretion in the stools together with the calcium and the fat intake. In the few cases in which fat values in the stools were obtained, there is shown the relation of the calcium excretion to that of total fat and fat as soap.

Calcium Excretion.—The range in calcium excretion was wider with rachitic than with normal children. There were seven values exceeding 1.0 gm. and the average, 0.86 gm., was higher than the normal average, 0.70 gm. In general, the excretion of calcium was related to the calcium intake, but not to the fat intake. In the few cases in which fat determinations in the stools were made the calcium excretion was found to be entirely unrelated to the amount of either total fat or fat as soap in the stools. In two instances there was a very large fat excretion in the stools and a very small calcium excretion; in three others there was a high calcium excretion, but only a small part of this excretion was in the form of soap.

TABLE 7.—Absorption of Calcium by Infants Suffering from Diarrhea

No.	Case	Age in Mos.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Per Cent. of CaO Intake Absorbed	Intake of Fat, Gm. per Kg.	Grams of CaO Intake per Gm. Fat Intake	Remarks
135	F. H. 4	11	4,838	Diarrheal	0.412	0.113	27.6	4.1	0.100	
109	S. J.	9	4,922	Severely diarrheal	0.362	0.063	17.4	5.9	0.061	Evaporated milk formula. Very large fermentative stools
132	F. H. 1	10	4,442	Severely dairrheal	0.348	0.059	16.9	7.3	0.048	
136	F. H. 2	10	4,660	Diarrheal	0.332	0.043	13.0	7.2	0.046	
175	I. G.	2.5	2,930	Diarrheal	0.304	0.021	6.7	5.0	0.061	Evaporated milk formula. Very large fermentative stools
106	V. C. 3	5	4,419	Diarrheal	0.282	0.138	49.2	6.8	0.041	Tetany, rickets, taking cod liver oil
121	V. C. 2	5	4,575	Severely diarrheal	0.248	0.059	23.9	5.2	0.048	Tetany, rickets, taking cod liver oil
120	V. C. 1	5	4,606	Severely diarrheal	0.234	0.062	26.6	5.1	0.048	Tetany, rickets, taking cod liver oil
143	F. S.	6	4,147	Mildly diarrheal	0.234	0.000	0.0	4.5	0.052	Considerable barley flour in formula
139	J. D. 2	5	3,930	Diarrheal	0.217	0.038	17.7	4.4	0.050	Tetany, rickets, taking cod liver oil. Magnesium sulphate injection
152	M. R.	4	3,001	Diarrheal	0.170	0.023	13.7	1.8	0.095	High proportion carbohydrate in diet
144	V. R. 2	3	6,357	Mildly diarrheal	0.154	0.038	24.5	2.9	0.052	Recently changed from breast milk
127	D. W. 4	5	5,724	Diarrheal	0.147	0.053	35.7	3.9	0.038	Rickets, tetany, taking cod liver oil
123	E. R. 3	11	7,367	Diarrheal	0.136	0.058	43.0	3.2	0.043	Rickets, tetany, taking cod liver oil. Low calories per kilogrom
147	W. H. 3	14	6,734	Diarrheal	0.134	0.071	53.3	2.7	0.049	Rickets, cod liver oil just discontinued
111	E. R. 2	10	7,436	Diarrheal	0.128	0.030	23.2	3.6	0.035	Rickets, tetany, taking cod liver oil. Low calories per kilogrom
131	E. R. 1	10	7,540	Diarrheal	0.123	0.000	0.0	2.2	0.055	Rickets, tetany, had castor oil. High proportion carbohydrate in diet
164	B. S. 1	12	8,105	Diarrheal	0.107	0.010	8.3	1.9	0.063	Rickets, tetany. Rather high carbohydrate in diet
176	B. S. 2	13	7,708	Severely diarrheal	0.095	0.004	4.1	1.5	0.063	Rickets, tetany. Rather high carbohydrate in diet. Excessively large stool

INFANTS SUFFERING FROM DIARRHEA

The third group of infants considered were suffering from diarrhea. Some of them were also rachitic, but are placed in this group because the diarrheal condition is the chief factor in determining the composition of the stools. Table 7 gives the absorption of calcium oxid per kilogram and its relation to the calcium and fat intake for these infants.

Calcium Absorption.—The range in intake of calcium per kilogram was very wide, and there were more high values than were found with normal infants. This was due largely to the fact that these infants were much under size. The absorption of calcium oxid was generally low. Only five of the nineteen values exceeded 0.06 gm. per kilogram; only two exceeded 0.09 gm. per kilogram. One of these two children had been receiving cod liver oil for about one week, and the other had a very high intake of calcium. The third highest absorption was in the case of a child who had been receiving cod liver oil just before the period of observation.

The average absorption for this group of infants was 0.046 gm. of calcium per kilogram, about one half that found for normal children.

The percentage absorption was also much below normal. Only four of the nineteen values fell within the normal range.

The fat intake per kilogram varied widely and bore no constant relation to the calcium absorption. Successive observations on two children, V. C. and E. R., showed that the calcium absorption increased as the diarrhea improved. Both these children were taking cod liver oil.

TABLE 8.—CALCIUM EXCRETION BY INFANTS SUFFERING FROM DIARRHEA

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap in Stools, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
109	S. J.	Severely diarrheal	1.78	29.1	1.47	14.54	1.29*	8.8*	7.3*
135	F. H. 4	Diarrheal	1.99	20.0	1.44	1.77	0.22*	1.5*	1.1*
136	F. H. 2	Diarrheal	1.54	33.6	1.34	4.61	0.87*	6.5*	5.6*
132	F. H. 1	Severely diarrheal	1.54	32.4	1.28	6.91	1.06*	8.3*	6.9*
143	F. S.	Mildly diarrheal	0.97	18.7	0.98	4.20	1.19*	12.2*	12.3*
131	E. R. 1	Diarrheal	0.93	16.8	0.94	3.35	0.00	0.0	0.0
164	B. S. 1	Diarrheal	0.97	15.5	0.89	3.07	0.00	0.0	0.0
121	V. C. 2	Severely diarrheal	1.13	23.6	0.86	10.38	0.34*	4.0*	3.0*
175	I. G.	Diarrheal	0.89	14.7	0.83	11.62	0.86*	10.4*	9.7*
120	V. C. 1	Severely diarrheal	1.09	23.6	0.80	5.77	0.00	0.0	0.0
144	V. R. 2	Mildly diarrheal	0.98	18.7	0.74	1.88	0.58*	7.8*	5.9*
111	E. R. 2	Diarrheal	0.95	27.1	0.73	3.76	0.00	0.0	0.0
139	J. D. 2	Diarrheal	0.85	17.0	0.70	6.01	0.82*	11.7*	9.7*
176	B. S. 2	Severely diarrheal	0.73	11.6	0.70	10.08	0.00	0.0	0.0
106	V. C. 3	Diarrheal	1.24	30.2	0.63	6.73	0.00	0.0	0.0
123	E. R. 3	Diarrheal	1.00	23.4	0.57	4.97	0.00	0.0	0.0
127	D. W. 4	Diarrheal	0.84	22.2	0.54	5.38	0.82*	15.2*	9.8*
152	M. R.	Diarrheal	0.51	5.4	0.44	3.61	0.13*	2.8*	2.6*
147	W. H. 3	Diarrheal	0.90	18.4	0.42	2.10	0.57*	13.7*	6.4*

* Value possibly too high. Stools acid.

Table 8 shows the calcium excretion and its relation to the calcium and the fat intake and to the excretion of total fat and of fat as soap in the stools of infants with diarrhea.

Calcium Excretion.—Both the calcium excretion and the calcium intake showed a wide range. The higher excretion usually occurred with the higher intake. The average excretion of calcium oxid was 0.86 gm. As in the other groups there was no definite relation between the fat intake and the calcium excretion. In some instances when the fat intake was high the calcium intake was also high, and the excretion of calcium higher than when the intake of calcium was lower. In this group, as in the others studied, there was no relation between the calcium excretion and the total fat excretion in the stools. Since the excretion of fat as soap in these acid stools was very low, evidently there was no relation between the excretion of calcium and that of fat as soap.

TABLE 9.—AVERAGE PROPORTIONS OF CALCIUM AND OF SOAP IN STOOLS OF INFANTS

Condition of Infants	Food	Stools	No. of Cases	CaO Intake, Gm. Daily	CaO, Percentage of Total Solids	CaO, Percentage of Total Salts	Fat as Soap, Percentage of Total Solids
Normal	Cow's milk	Constipated	13	1.21	11.1	49.6	30.0
Normal	Cow's milk	Normal	19	1.23	11.8	47.5	24.5
Nearly normal	Cow's milk	Softnormal	7	1.06	10.0	46.3	17.7
Mostly delicate	Cow's milk	Nonhomogeneous	13	1.02	7.9	40.9	13.6*
Sick	Cow's milk	Diarrheal	13	1.05	6.3	32.5	2.8*
Sick	Cow's milk	Severely diarrheal	6	1.12	4.4	25.2	2.8*
Rachitic	Cow's milk	Constipated or normal	9	1.37	12.7	44.7	15.7*†
Rachitic	Cow's milk	Softnormal or nonhomogenous	14	0.94	8.1	39.1	15.5*‡
Normal	Breast milk	Normal	3	(0.38)§	3.4	37.4	23.4*
Normal	Breast milk	Nearly normal, partly green	12	(0.38)§	4.3	42.9	12.0*
Normal	Breast milk	Green, with mucus	5	(0.38)§	3.0	39.6	4.2*
Sick	Breast milk	Diarrheal	4	0.22	1.8	18.4	4.6*
M. I., normal	Top milk, cream dilution	Normal	1	0.27	2.1	28.0	50.6
C. W., normal	Malted milk	Softnormal	1	0.26	7.5	40.6	11.0

* Value possibly too high. Stools acid.
† Only two values included in this average.
‡ Only three values included in this average.
§ Average of five values for normal children.

PROPORTION OF CALCIUM AND OF SOAP IN THE STOOLS OF INFANTS

Table 9 gives the average proportion of calcium in various types of stools of infants and its relation to the other constituents of the stools and to the calcium intake. There are given values not only for stools of infants taking modifications of cow's milk, but also for a number of stools of breast fed infants.

The stools of infants taking modifications of cow's milk which contained the smallest proportion of water, that is, the constipated and the normal stools, showed on the average the higher calcium percentage of total solids. With increased proportion of water the calcium percentage of total solids became less. The total calcium excretion was not thereby diminished, however, but rather increased, since the daily amount of total solids was greatly increased with the higher water content. The calcium percentage of total solids appeared to be more closely related to the looseness of the stools than to the amount of calcium intake, since there was but little variation in average intake for the different groups.

The calcium percentage of total solids of the constipated or normal stools of rachitic infants was higher than that of the corresponding type of stools of normal infants. It is not evident whether this increased percentage was due to the condition of rickets or to the higher average intake.

The average values for calcium percentage of total *salts* also were less as the stools became more watery. In other words, the proportion of salts not calcium, that is, the soluble salts, increased with the water content of the stools.

In stools of rachitic infants, although the total salts were increased over the normal, the calcium formed a smaller percentage of the total salts than in the corresponding stools of normal children. This shows that the other salts were increased more than was the calcium.

The proportion of the total solids which was soap in the stools of infants taking modifications of cow's milk was closely related to the water content, diminishing strikingly as the water in the stools increased. In the looser types of stools the decrease in soap percentage with increase in water content was accompanied by a similar though less marked decrease in the proportion of calcium. This association was not found in the constipated and the normal stools. The normal stools showed a lower proportion of fat as soap, with a slightly higher percentage of calcium, than did the constipated stools.

An entirely different range of values was seen with breast fed infants. With a much lower intake of calcium, the calcium percentage of total solids in the stools was much lower than when the food was modifications of cow's milk. The diarrheal stools of breast fed infants showed a much lower calcium percentage of both total solids and total salts than did the other types of stools. The other groups of the stools of breast fed infants did not differ much from one another in water content and varied little in the calcium percentage of total solids or of total salts. The calcium formed a somewhat smaller part of the total salts than in the stools of artificially fed infants.

The fat as soap, as well as the calcium oxid, formed a smaller proportion of the total solids in the stools of breast fed infants than in those of infants taking cow's milk, and was less in the diarrheal than in the normal stools. In the three groups of stools of breast fed infants which were normal as to water content, the proportion of soap was lower the less normal the appearance of the stool, while the calcium percentage of total solids varied but little.

The proportion of calcium in the stools in two instances of unusual feeding, with extremely low calcium intake, are included in Table 9. In the first instance, in which the food was a cream dilution, the calcium percentage of both total solids and total salts was very low, even lower than that found with breast fed infants. This stool had a very high percentage of soap, due to the administration of a large amount of milk of magnesium. In the other instance, in which the child received malted milk, the calcium percentage of total solids and of total salts was somewhat lower than that found for the same type of stool when the food was a modification of cow's milk.

The following summary gives answers, based on our observations, to such of the questions stated in the introduction as apply to infants taking modifications of cow's milk.

SUMMARY

1. The average absorption of calcium oxid by healthy infants taking modifications of cow's milk was 0.09 gm. per kilogram of body weight. Since the average absorption of calcium oxid by breast fed infants was about 0.6 gm. per kilogram, it may be assumed that 0.06 gm. per kilogram is the minimum normal absorption by infants taking modifications of cow's milk.

The daily total excretion of calcium oxid in the stools ranged from 0.34 to 1.06 gm., averaging 0.70 gm.

2. The excretion and the absorption of calcium were, in general, dependent on the amount of calcium intake, from 35 to 55 per cent. of the intake being absorbed.

To insure the average absorption of 0.09 gm. of calcium oxid per kilogram, the intake of calcium oxid should be at least 0.19 gm. per kilogram; to insure an absorption equal to the average found for breast fed infants the intake of calcium oxid should be at least 0.13 gm. per kilogram.

The best absorption of calcium was obtained when the calcium intake bore a definite relation to the fat intake, that is, when the food contained from 0.045 to 0.060 gm. of calcium oxid for every gram of fat and when at the same time the fat intake was ample, not less than 4.0 gm. per kilogram.

3. An excessive calcium intake apparently did not increase the calcium absorption, the excess being excreted. When the intake of calcium oxid was very low, less than 0.10 gm. per kilogram, the absorption of calcium oxid was less than the normal calcium requirement of the body.

4. The *total* absorption of calcium oxid varied in general with the weight of the child; the *per kilogram* absorption did not vary regularly with either the age or the weight.

5. The relation of calcium excretion to soap excretion was not constant. The excretion of *soap* was directly related to the type of stool, that is, to the water content and to the reaction of the stool. The excretion of *calcium* was closely related to the calcium intake. On the average, the normal and the constipated stools, with high soap content, were found when the intake of calcium was high and, therefore, they showed the higher excretion of calcium. However, constipated stools, which contained more soap than normal stools, had lower calcium content. Nonhomogeneous stools, with the lowest average soap content, showed the same content of calcium not held as soap as did the constipated stools, with the highest soap content.

6. The calcium that could be lost as soap was never a large proportion of the calcium intake. Even in the stools containing the most soap it was found to be less than three-tenths the calcium intake. The calcium lost as phosphate was shown not to be increased in soapy stools.

7. The calcium percentage of the total solids varied, as a rule, with the water content of the stools, diminishing as the water increased.

8. The calcium absorption was much lower when diarrhea was present. With an increased excretion of calcium in diarrheal stools, there was a marked decrease in soap excretion.

The calcium absorption by rachitic infants was much lower than that by healthy infants.

In the few cases in which observations were made on infants recovering from rickets, the calcium absorption was higher than the normal average. These infants had received cod liver oil for a considerable period.

9. The administration of cod liver oil regularly increased the absorption of calcium, unless diarrhea was present.

INCIDENCE OF PROTEIN SENSITIZATION IN THE NORMAL CHILD

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The object of this paper is to show the incidence of protein sensitization in the normal child. Subsequent papers will show the incidence of sensitization in asthmatic and eczematous patients in comparison with the normal.

This study of protein sensitization in its relationship to the diseases of infancy and childhood as manifested in eczema, asthma and urticaria, has been made possible through alumni of this institution.

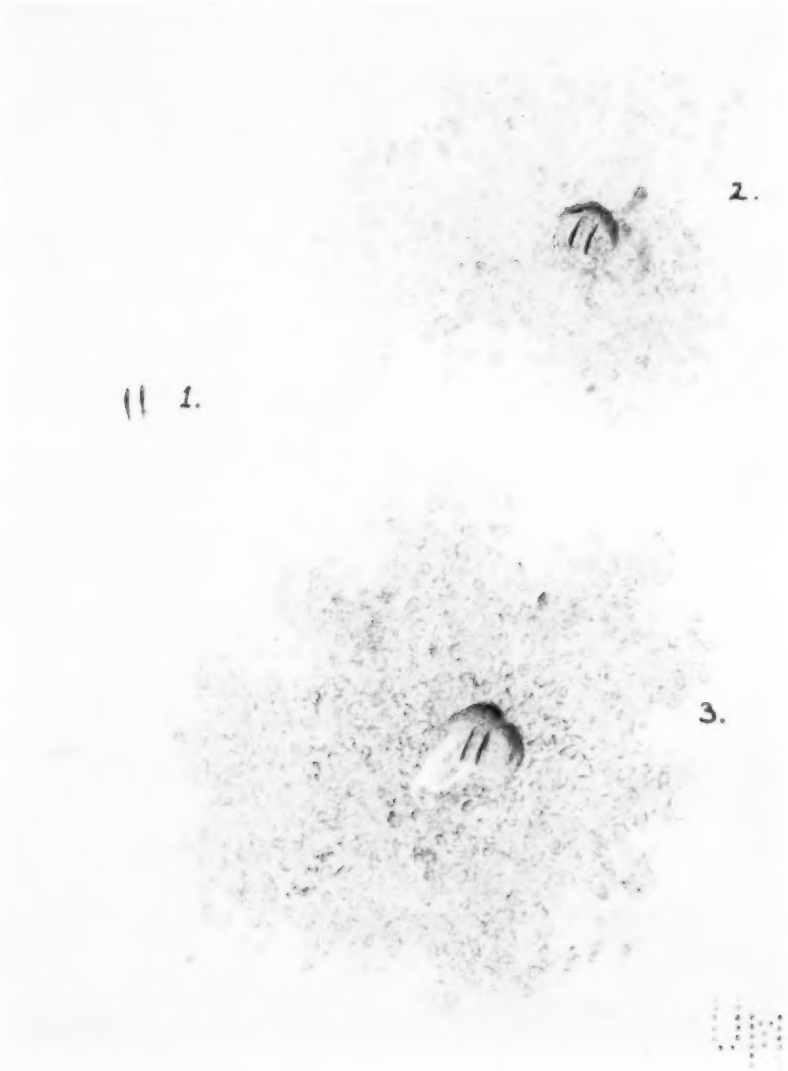
This research has necessarily been brought to a standstill, before definite conclusions could be reached because of the war, but the results thus far are very suggestive and of interest to the pediatrician to note the striking difference between the incidence of sensitization in the normal as compared with the child showing anaphylactic symptoms.

Within a few years, numerous reports have appeared in regard to the treatment of hay-fever and asthmatic patients by determining the presence or absence of protein sensitization. In all of these reports there have unquestionably been sufficient controls to check the relationship, but it has seemed to me in studying the problem of protein sensitization, that it is highly important to have as control a series of tests in normal cases, which will determine (1) the incidence of sensitization in the normal child; (2) show the relative frequency of sensitization to the specific proteins; (3) give a standard by which results of the reactions on anaphylactic patients may be determined properly.

To carry out this test for the detection of hypersusceptibility toward various proteins, the preparation of extracts are of fundamental importance. In order to get the best results it is essential that the preparations used should readily be held in suspension. They should be sterile and suspended in a non-irritating and readily absorbable diluent.

In the preparation of the protein extracts, the first obvious step is to avoid the use of chemicals which may form insoluble compounds with the proteins present or form new compounds which might lead to erroneous results. Consequently, the proteins of vegetables and fruits have been prepared as described by Woodhouse. The methods principally used were described in a previous study by Baker and Floyd on protein extracts in states of hypersensitization.

All proteins employed in this study were used on anaphylactic as well as on normal patients, so that a comparison can be made between the patients giving no anaphylactic symptoms and those with varying degrees of sensitization.



Types of reactions in skin sensitization test: 1. Control, no reaction. 2. moderate reaction. 3. Severe reaction.

Illustrating Article by Horace Mitchell Baker, M.D.
American Journal of Diseases of Children, February, 1920

The method employed to determine sensitization was carried out in the following manner: The inner side of the forearm was cleaned with alcohol and ether, scarifications or scratches were made about two inches apart, allowing the oozing of serum only. The upper scratch was kept for a control. One or two drops of the protein in normal saline or tenth normal sodium hydroxid are placed on the other scratches and allowed to be absorbed. Both points of inoculation are examined at five minute intervals for half an hour.

Positive reactions consist of a raised urticarial wheal, surrounded by an area of redness of the skin, irregular in outline and usually from 6 to 20 mm. in diameter. The reactions described as questionable consist either of a slight elevation about the point of inoculation or of a small area of erythema. Such reactions are considered negative for diagnostic purposes. They are noted and repeated at a later time in that they may be positive or entirely negative on the subsequent examination. Negative reactions are those which do not differ from the control.

The number of positive, doubtful and negative reactions with each protein is shown.

TABLE 1.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Vegetables

	Number Tested	Number Positive	Questionable Number	Number Negative
Bean	24	0	0	24
Beet	24	0	0	24
Cabbage	24	0	0	24
Cucumber	24	0	0	24
Lettuce	24	0	0	24
Tomato	24	0	1	23
Onion	24	1	0	23
Parsnip	24	0	0	24
Peas	24	0	0	24
Potato	24	1	0	23
Squash	24	0	1	23
Spinach	24	0	1	23
Turnip	24	0	0	24

TABLE 2.—Cases Giving History of Anaphylaxis Tested for Sensitization to Vegetables

	Number Tested	Number Positive	Questionable Number	Number Negative
Beet	4	0	0	4
Bean	30	1	1	28
Spinach	15	1	3	11
Cabbage	18	0	0	18
Tomato	12	0	0	12
Carrots	14	0	0	14
Cucumbers	16	0	0	16
Lettuce	16	1	0	15
Potato	34	7	2	25
Parsnip	23	1	0	22
Peas	30	3	0	27
Squash	26	2	2	22
Turnip	17	1	0	16
Onion	22	1	2	19

TABLE 3.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Eggs and Milk

	Number Tested	Number Positive	Questionable Number	Number Negative
Egg white	25	1	0	24
Whole egg	25	0	0	25
Casein	25	0	0	25
Lactalbumin	25	0	0	25

TABLE 4.—Cases Giving History of Anaphylaxis Tested for Sensitization to Eggs and Milk

	Number Tested	Number Positive	Questionable Number	Number Negative
Eggs	74	6	6	62
Casein	76	4	0	72
Lactalbumin	51	0	1	50

TABLE 5.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Fish

	Number Tested	Number Positive	Questionable Number	Number Negative
Clam	25	0	0	25
Codfish	25	0	2	23
Haddock	25	0	1	24
Lobster	25	0	1	24
Salmon	25	4	1	20
Oyster	25	0	0	25

TABLE 6.—Cases Giving History of Anaphylaxis Tested for Sensitization to Fish

	Number Tested	Number Positive	Questionable Number	Number Negative
Codfish	33	0	0	33
Haddock	30	0	3	27
Salmon	25	0	3	22
Lobster	32	0	1	31
Clam	30	0	0	30
Crab	23	0	0	23
Oyster	28	0	1	27

TABLE 7.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Grains

	Number Tested	Number Positive	Questionable Number	Number Negative
Buckwheat	25	0	2	23
Barley	25	0	0	25
Corn	25	1	0	24
Oats	25	0	0	25
Rice	25	0	0	25
Rye	25	1	0	24
Wheat	25	0	0	25

TABLE 8.—Cases Giving History of Anaphylaxis Tested for Sensitization to Grains

	Number Tested	Number Positive	Questionable Number	Number Negative
Wheat	48	1	2	45
Oats	69	9	4	56
Rye	37	0	3	34
Barley	35	0	3	32
Rice	65	4	3	58
Corn	40	2	3	35
Buckwheat	26	0	1	25

TABLE 9.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Meats

	Number Tested	Number Positive	Questionable Number	Number Negative
Beef	25	0	0	25
Chicken	25	0	0	25
Lamb	25	0	0	25
Pork	25	0	0	25
Veal	25	0	2	23

TABLE 10.—Cases Giving History of Anaphylaxis Tested for Sensitization to Meats

	Number Tested	Number Positive	Questionable Number	Number Negative
Beef	50	3	2	45
Lamb	48	2	0	46
Veal	33	0	0	33
Pork	36	0	0	36
Chicken	45	3	2	40

TABLE 11.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Nuts

	Number Tested	Number Positive	Questionable Number	Number Negative
Almond	11	0	0	0
Peanut	22	0	0	0
Brazilnut	17	0	0	0

TABLE 12.—Cases Giving History of Anaphylaxis Tested for Sensitization to Nuts

	Number Tested	Number Positive	Questionable Number	Number Negative
Peanut	17	0	0	17
Almond	14	0	0	14
Brazilnut	17	1	1	15

TABLE 13.—Cases Giving No History of Anaphylaxis Tested for Sensitization to Fruits

	Number Tested	Number Positive	Questionable Number	Number Negative
Banana	25	0	0	25
Grapefruit	25	0	0	25
Strawberry	25	1	1	23
Orange	25	0	0	25

TABLE 14.—Cases Giving History of Anaphylaxis Tested for Sensitization to Fruits

	Number Tested	Number Positive	Questionable Number	Number Negative
Banana	16	0	1	15
Orange	10	0	0	10
Grapefruit	16	0	2	14
Strawberry	21	2	3	16

TABLE 15.—Summary of Positive Reactions in the Normal Control Cases

	Positive Reactions	Questionable Reactions	Total Positive and Questionable
Onion	1	0	1
Squash	0	1	1
Spinach	0	1	1
Potato	1	0	1
Egg white	1	0	1
Salmon	4	1	5
Codfish	0	2	2
Haddock	0	1	1
Lobster	0	1	1
Corn	1	0	1
Rye	1	0	1
Buckwheat	0	2	2
Veal	0	2	2
Strawberry	1	0	1

These articles of diet which give a positive or questionable reaction in apparently normal cases is significant in that it includes many of the common articles of diet which pediatricians usually avoid in regulating diets of children, because of experience, that they cannot be tolerated. The presence of sensitization in the absence of symptoms, may be due to a low degree of sensitization not sufficient to cause anaphylactic manifestations.

Thus it is evident from these tables that the incidence of sensitization of apparently normal children is almost a negligible factor, except in the case of salmon, which reaction in any case cannot be considered as an absolute indication of sensitization. The articles of diet most commonly causing disturbance in children presenting anaphylactic symptoms are:

1. Oatmeal
2. Potato
3. Eggs
4. Peas
5. Rice
6. Casein
7. Beef Juice
8. Chicken

This does not mean that a child with anaphylactic symptoms is not capable of taking any one or all of these articles of diet. Only a careful examination can reveal the causative factor. In many instances it is only one food while it may be several.

It is only by the most careful study and examination of the case, with a definite knowledge of reading the specific reactions, that the form of sensitization can be determined.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES*

EDWIN E. GRAHAM, M.D.
PHILADELPHIA

During the past two years, through the courtesy of Dr. Chevalier Jackson, I have had an opportunity of seeing in children a comparatively large number of cases of foreign bodies in the air passages, and a much smaller number of cases of foreign bodies in the food passages. I feel much indebted to Dr. Jackson for the opportunity of studying these cases, as all of them were referred to him, and he later referred them to me for medical examination as to the location of the foreign body and for medical treatment.

The subject has been of such great interest to me, and has taught me so much that I feel it may interest you if I draw attention to some of the points that seem to me most important in connection with these foreign body cases.

Of the comparatively large number of cases that I have been seeing, I have selected some of the most interesting. A brief summary of the clinical history of four of these cases will suffice to illustrate their nature.

REPORT OF CASES

CASE 1.—James W. F., aged 4 years, was admitted to Jefferson Hospital, April 19, 1919. Diagnosis: Carpet tack in right lung. Location of foreign body: lower lobe of right lung. Tack aspirated, March 15, 1919. Two previous unsuccessful bronchoscopies. Foreign body removed by Dr. Chevalier Jackson, April 23, 1919. Time: thirty-five minutes, ten seconds. Uneventful recovery. Discharged, April 26 (Fig. 1).

CASE 2.—Frank S., aged 2 years, was admitted to Jefferson Hospital, March 19, 1919. Diagnosis: Peanut kernal in right bronchus. Location of foreign body: right bronchus at orifice of upper lobe bronchus. Peanut aspirated, March 16, 1919. Symptoms: Marked wheezing, cyanosis, severe cough, fever. Tracheotomy: March 20, 1919. Kernel removed by Dr. Chevalier Jackson, March 25. Time: four minutes, twenty-six seconds. Recovery (Fig. 2).

CASE 3.—William B., aged 4 years, admitted to Jefferson Hospital, Dec. 20, 1917. Diagnosis: Staple in left bronchus. Location of foreign body: in left main bronchus. Staple aspirated, Dec. 14, 1917. Foreign body removed by Dr. Chevalier Jackson, Jan. 8, 1918. Time: eighteen minutes, fifty seconds. Recovery. Discharged, January 11 (Fig. 3).

CASE 4.—Nathaniel M., aged 8 years, admitted to Jefferson Hospital, Jan. 8, 1919. Diagnosis: Cartridge blank in left bronchus. December 24, patient aspirated empty cap of a 22 caliber bullet. Location of foreign body: at orifice of left upper lobe bronchus. Foreign body removed by Dr. Chevalier Jackson, January 11. Time: eight minutes, thirty-one seconds. Recovery (Fig. 4).

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* President's address, read before the American Pediatric Society, Atlantic City, N. J., June 16, 1919.

It seems only plausible to believe that if one man can see more than seven hundred foreign body cases, and this has been the experience of Dr. Jackson, that very many foreign body cases terminate in death without a correct diagnosis being made, and without any attempt at removal of the foreign body by endoscopy.

The period of latency of symptoms which follows the primary violent dyspnea and choking attack, and later the gradual onset and chronic character of the symptoms suggest to the clinician some acute or chronic pulmonary disease, consequently one not accustomed to the study of these cases might fail to suspect the presence of a foreign body in the lung.

When one reads the histories of these children, their being referred from one competent physician to another, one is struck by the fact that in spite of the history given by the parents of having swallowed or inhaled a foreign object, little attention is paid to this fact, and very often a roentgenogram of the chest is not taken. One of the important lessons I have learned in the last two years from the study of these cases is to suspect a foreign body in the lung, if the following conditions are present: localized lung symptoms that persist in spite of treatment, no tubercle bacilli in the sputum, a leukocytosis for which there seems no definite reason, and a gradual failure in health and weight.

Foreign bodies in the lungs have been regarded practically as curiosities. So far as I can remember only one paper has been read before this society on this subject, and that some years ago. In it, if I remember correctly, three cases were reported, and in the discussion I reported two or three cases that I had found accidentally.

The symptoms of a foreign body in the air passages vary largely, depending on the nature of the foreign body. The peanut kernel immediately sets up a violent dyspnea and an accumulation of purulent secretion in the lungs due to the severe laryngitis, tracheitis, and bronchitis. On the other hand, the inhalation of a metallic foreign body, such as a tack, produces for a very short time violent dyspnea and choking after which there is a period of quiescence in which the patient is entirely free from symptoms for weeks or even months.

The period of quiescence is followed by gradually failing health and strength, cough, moderate fever and a clinical picture that suggests incipient tuberculosis. Undoubtedly foreign body cases have often been so diagnosed.

Metal objects, if they block a bronchus completely, cause a retention of the secretions and may quickly form an abscess. If, however, they occlude the bronchus only partially and the secretions can be coughed up, they may remain for a very long time and do comparatively little damage. The swelling of the mucous membrane, which is always

associated, is a factor which ultimately assists greatly in the blocking up of the secretions.

Foreign bodies in children are associated with a large amount of secretion. This excess of secretion is probably due both to the fact that the child's lung is more susceptible to the presence of a foreign body than the adult's is, and the fact that the child cannot, like the adult, remove the secretion by coughing. Statistics tend to show that about 66 per cent. of the cases of foreign bodies in the air passages occur in children, due, in my opinion, to the fact that children are very apt to put any and all objects into the mouth and exercise less care in removing them from the mouth than does the adult.

If in the history of the case there is no such history of dyspnea and choking, and one is sure that these symptoms were never present, it is a strong argument against the inhalation of a foreign body.

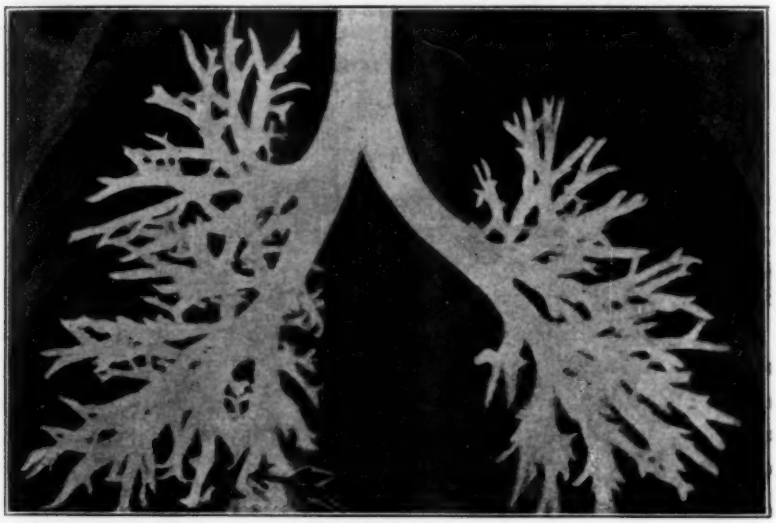

Fig. 1.—Carpet tack in lower lobe of right lung.

Food substances, such as bread, lean or fat meat, or those that are apt to be more or less quickly dissolved are usually coughed up and, as a rule, produce no dangerous symptoms. Fine particles of dust are ordinarily coughed up, but large particles, such as coal dust, often become encysted, producing anthracosis. The larger bodies may and probably do produce many of the pulmonary abscesses that are non-tuberculous.

In all foreign body cases a physical examination of the child's entire body, especially of the chest, is most important. Some foreign bodies do not cast a shadow on a photo-sensitized plate, and the study

of a large number of such cases ought to be of value. The lung symptoms are nearly always most marked in the lung containing the foreign body. The study of the physical signs, both before and after bronchoscopy, is of importance. The large amount of secretions sometimes removed by the bronchoscopy may cause the signs of consolidation in a lung to disappear. This accumulation of secretions is apt to occur in the lower lobe of the lung, and from a study of a number of such cases it is evident that the secretions gradually accumulate in this lower lobe by gravity, this accumulation taking place in spite of efforts to remove it by coughing.

If a foreign body is present in the esophagus, food may be swallowed easily and regurgitated, or swallowed with difficulty, as is the case in stricture of the esophagus. This is probably due to an associated spasm of the esophagus in the majority of cases, or less often to the change in the position of the body in the esophagus. Sometimes it is impossible to decide on the location of the foreign body in the esophagus from the sensations of the patient. Foreign bodies in the larynx usually produce hoarseness, with a croupy cough and evidences of severe dyspnea. This signifies either glottic or subglottic lodgment.

The asthmatoid wheeze which may be heard in some of the foreign body cases, is, when present, a symptom of considerable importance, especially in those cases where the foreign body does not show a shadow on the plate. It is heard during or at the end of expiration, by placing the ear or stethoscope in front of the mouth of the patient.

It is ill advised to urge a patient to cough with the hope of expelling the object inhaled. The possibility of his coughing it up is very slight, and the probability of his doing injury by forcing the sharp point of the object inhaled, such as a tack, into the laryngeal mucosa is very great.

A larger number of foreign bodies enter the right than the left bronchus, from 62 to 75 per cent. according to different observers. Consequently more foreign bodies lodge in the right lung than in the left, and they rarely lodge in the middle lobe bronchus. There are good anatomical reasons for this, as the diagram from Dr. Jackson's book clearly shows (Fig. 5):

The right bronchus is larger than the left, and the angle of deviation is less acute; and the carina, i. e., the top of the bifurcation, is to the left of the middle line of the trachea.

Heavy objects, owing to their weight in comparison to their small size, are rarely coughed up. In fact, very light substances, such as cork, are rarely spontaneously removed by coughing, and the consensus of opinion today is to remove the foreign body by endoscopy.

The physical signs of foreign bodies that have localized themselves in the lung depend on the amount and extent of the lesions. They may vary according to the composition, form, shape and size of the foreign body, as to whether it blocks the bronchus slightly, largely or completely, and also, to a certain extent, on the amount and character of the secretions. There may be a simple local bronchitis, slight or severe local congestion, slight or much retained purulent secretions, bronchiectasis, or pulmonary abscess. When the foreign body has been present for a long period of time, there is usually a moderate degree of bronchitis in the opposite lung, probably due to the retained secretions gaining access to the other lung, but with the exception of the peanut and maize cases, the local evidences around the foreign body are so well marked that they are usually easily recognized, espe-

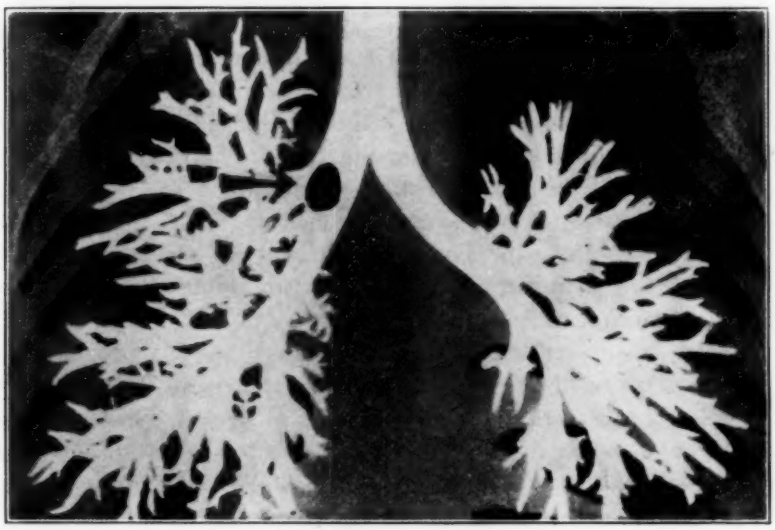

Fig. 2.—Peanut kernel in right bronchus.

cially in those cases where the foreign body has been present long enough to produce distinct local pathology. In a case where a foreign body has been present in the lung for a long period of time there is usually considerable cough with offensive expectoration.

If a cavity forms, it may be either a bronchiectatic or an abscess cavity, and there is an absence of tubercle bacilli in the sputum. In a recent case the expectoration is less offensive and more of a muco-purulent character, and occasionally streaked with blood.

Dr. Jackson speaks often of "drowned" lung in his foreign body cases. In a number of cases it is noticed that a great deal of opacity in the tissues surrounding the foreign body disappears after bronchos-

copy. This is evidently due to the copious outpouring of secretions after the bronchoscopy. In some of the cases where the foreign body has been present for a number of years, some evidences of damage to the lung may still be visible in the roentgenogram months after the object has been removed, although at this period all physical signs have disappeared clinically. A "drowned" lung, according to Dr. Jackson, is caused by the drainage becoming blocked by the foreign body. The passages of the lungs become filled with secretions, which collect easily, and these consequently become purulent. If this area of drowned lung is not relieved by the removal of the foreign body from the bronchus, we have finally an abscess cavity.

Foreign bodies are occasionally coughed up. It is more likely to be coughed up if the object is in the larynx than if it is in the bronchi. The possibilities of a foreign body being coughed up are very slight, however. The nature of the foreign body has something to do with it. Pins and sharp bodies are rarely if ever expelled by coughing, as the effort of coughing causes the sharp point to become more tightly fixed. Round, smooth objects usually become firmly lodged, the air below the object is absorbed and coughing consequently can hardly ever be strong enough to disturb the foreign body, as little air is below it. Heavy bodies, as stated before, are seldom coughed up.

According to the experience of Dr. Jackson, pins are the most common foreign bodies in the bronchi, next in frequency are different kinds of hardware, then vegetable substances.

If there is any reason to suspect a foreign body, a bronchoscopy should be performed. It is important to appreciate that a foreign body does not necessarily produce any physical signs or symptoms, and, on the other hand, it is important to remember that certain lesions in the air passages, as a false membrane, may closely simulate the physical signs and symptoms of a foreign body.

In a case of suspected bronchiectasis, a bronchoscopy may assist in clearing up the diagnosis, especially if the symptoms are limited to one base and are suggestive of tuberculosis, but no tubercle bacilli are found.

There are no contraindications to bronchoscopy, except, perhaps, extreme weakness of the patient, when time should be given the individual to rally partially. Pulmonary abscess, pneumonia, pulmonary gangrene or status lymphaticus, are not contraindications for bronchoscopy.

Bronchoscopy should be performed as soon as possible after the entrance of the foreign body. The longer the time that elapses, the more difficult is the removal owing to the swelling and edema of the bronchial mucosa, later the formation of granulation, and also the

fact that the natural tendency of the foreign body is to sink deeper and deeper into the lung. The child's health and strength gradually become impaired, which, of course, tends to make convalescence more tedious.

I have never seen Dr. Jackson give an anesthetic to children, although we have occasionally given a small dose of morphin with atropin to older children before the bronchoscopy.

The necessity of making a roentgenogram in every case in which a history of having swallowed or inhaled a foreign body is given cannot be emphasized too strongly. If a suspicion exists that a foreign body may be present, a roentgenogram should be made, or if an unexplained leukocytosis is present, connected with local physical

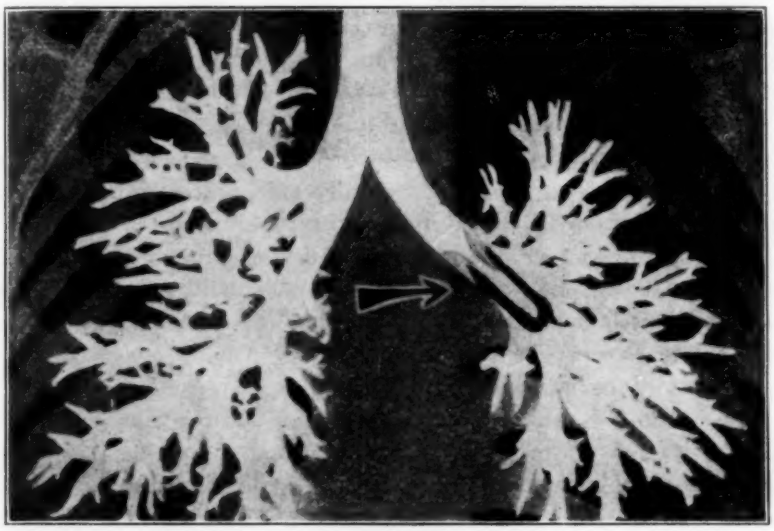

Fig. 3.—Staple in left bronchus.

signs in the lungs that do not clear up under treatment, and if there are no tubercle bacilli in the sputum, the necessity for taking a roentgenogram is evident. It is difficult to explain why so many patients with definite local lesions in one lung that are nontuberculous and that do not disappear under treatment, are not given the benefit of roentgen-ray examination. The probable explanation is that the medical profession, as a whole, does not appreciate the frequency of foreign bodies in the lungs, and the history of havng swallowed or inhaled a foreign body is disregarded owing to the period of latency which exists between the time of inhalation and the first development of symptoms.

In a roentgenogram one side may be dark and the other side light. The dark side usually contains the foreign body, perhaps occluding a

large bronchus, which may produce a well marked emphysema in the unobstructed lung. It is well to remember that calcified glands may show a rounded shadow.

It is important to appreciate that a negative roentgenogram does not absolutely prove that a foreign body is not present. Metal objects may not show, or the object may not be opaque to the roentgen ray. The physical signs in such a case are often a valuable guide.

The location of a foreign body in the esophagus that shows but slightly, if at all, in the roentgenogram may be facilitated by having the patent swallow a capsule containing bismuth. This capsule may lodge just above the foreign body. It is important to remember that in a perfectly free esophagus the capsule may stop for a moment in its passage through the esophagus.

If the foreign body is in the lung, the bronchoscope, and if in the esophagus, the esophagoscope is used. Both are passed through the mouth. The illumination is at the distal end, and all the work is done by sight; nothing is done in a blind manner.

The extraction of foreign bodies of steel or iron by the magnet has very narrow limitations, practically only such bodies can be removed as are easily removed by bronchoscopic means.

The bronchi do not diminish in size between the branches. The lessening in the lumen occurs at the point of subdivision between the branches. The lumen between the branches is of a uniform size and does not taper, consequently a foreign body usually is checked and stops below where branching begins.

The diagnosis is made from the history of a foreign body being in the mouth, with an attack of violent dyspnea, and choking sensations. Except when substances such as a peanut kernel or maize is inhaled, this is followed by a period of quiescence and freedom from all symptoms that often lasts for weeks or months. The physical signs are largely localized to a comparatively small area in one lung.

As regards prognosis, Dr. Jackson reports 98.1 per cent. successful removals. His mortalty during operation is less than 0.5 per cent. Including all deaths within one month after endoscopy in more than 700 cases, his mortality is 1.9 per cent. Even this low mortality, if studied carefully, will be found to be due almost entirely to the fact that the patients either were almost moribund when the attempt to remove the foreign body was made, or else the object had been present in the lung so long that notwithstanding its removal the patient later succumbed to the lung condition.

In Jackson's twenty-three cases where the foreign body had been present for from two months to twenty-six years, there were two deaths. The recovery from the pulmonary conditions present is, as a

rule, complete, as far as symptoms are concerned. Cough, expectoration and fever disappear, the child gains in weight and strength, and recovery is apparently complete. The later roentgenologic study of the lungs in these cases also shows a remarkable and to the clinician often an unexpected recovery.

In regard to prophylaxis, all children should be prevented as far as possible from putting any object in the mouth, and when old enough they should be taught the danger of putting objects in the mouth. Great care should be taken that no foreign body gains access to the food before it is eaten. Bones, pins and numerous small objects, such as china and glass, are found in foodstuffs, and as a result may be swallowed or inhaled, if not noticed during mastication. It is most

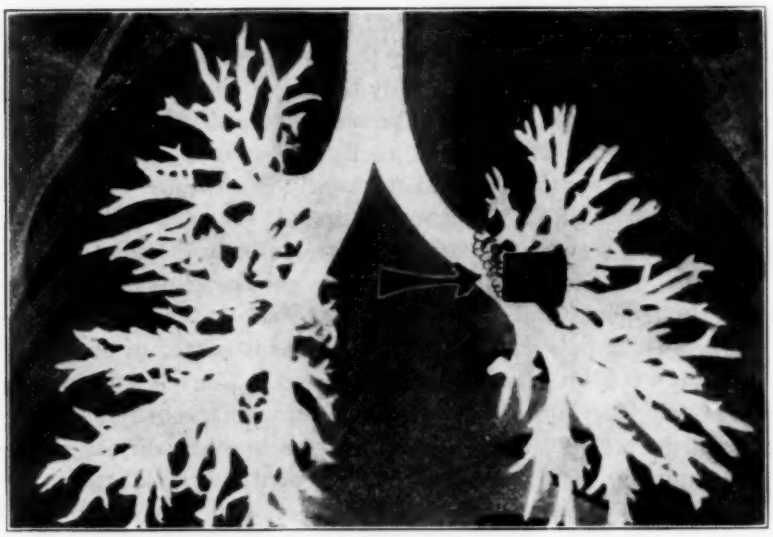

Fig. 4.—Cartridge blank in left bronchus.

important not to leave objects small enough to be placed in the mouth lie where young children can pick them up. A good sized safety pin is not too large to be placed in the mouth of the young child, and possibly swallowed or inhaled.

In children less than 2 years of age a bronchoscopy should not last more than thirty minutes, unless it is urgently required to prolong it, when it may last for one hour. Children more than 2 years of age will easily stand a bronchoscopy lasting one hour. This is done without either general or local anesthesia, although occasionally a small preliminary dose of morphin and atropin is given. Many of Dr. Jackson's operations for removal of foreign bodies are done in two or three minutes.

As regards treatment, with an early roentgen-ray examination and a bronchoscopy, the treatment is most satisfactory. If a foreign body is inhaled, the patient should be kept as quiet as possible, and as much in the recumbent position as possible, until after the larynx has been examined by the mirror and a roentgenogram has been taken. The idea in keeping the patient recumbent is to keep the foreign body from gravitating lower into the lung. It is important to quiet the patient as much as possible, and he should never be urged to cough with the idea of possibly coughing up the foreign body. It is never wise to pass a bougie into the esophagus for the location of a foreign body or where disease is present. The passage of the bougie practically gives us very little knowledge, and the passage of the esophagoscope, which is certainly no more difficult, gives us valuable knowledge. As a second "don't" to the passage of the bougie might be mentioned the fact that the child should never be held up by the heels and slapped on the back, as there is danger of the foreign body becoming detached, and asphyxiation may result from the object becoming caught in the glottis. Of course, it goes without saying that in all cases of foreign body a roentgenogram should be taken, and taken as early as possible.

All children should be kept in bed and, if possible, under a competent nurse for several days after the foreign body is removed.

As a rule, there is little or no fever after the bronchoscopy, although at times the temperature rises to 100 F. Usually, even in these cases, it falls to normal in a day or two. This was a matter of surprise to me in the first few bronchoscopies that I saw Dr. Chevalier Jackson do, as I expected fever and cough to follow the passage of the bronchoscope, and the manipulation with the instruments necessary to remove the foreign body. The instruments used are, of course, all sterile, and the sensitiveness of the deeper bronchi is very slight as compared with the larynx, hence the child stands the necessary intrusion of the deeper structures well and there can be no infection, as no local or constitutional symptoms follow the operation. If pneumonia is present before the bronchoscopy there may be a severe reaction, although in none of such cases that I have seen has it been fatal.

If before the bronchoscopy, a bronchitis, tracheitis or laryngitis with fever is present, these symptoms are usually aggravated temporarily by the bronchoscopy. The reaction was most marked in the peanut cases. If the epithelium is injured either by the foreign body or by the necessary manipulation, the reaction may be severe following the passage of the bronchoscope. A moderate degree of hoarseness is often seen in children, which usually lasts only two or three days. What Dr. Jackson speaks of as a "drowning of the patient by his own secretions" has been noted in a number of cases in children, especially

in the peanut cases. This is usually relieved by the passage of the bronchoscope, and the "sponge pumping" process.

Subglottic edema may be present before the bronchoscopy, due in some cases to the presence of a foreign body in the subglottic region, or in the trachea, or it may be caused by the cough or respirations moving the foreign body backward and forward. Jackson believes that distal illumination and the use of small tubes lessens the risk of the production of this edema of the glottis. If the symptoms are due to subglottic edema a low tracheotomy gives great relief, as I have seen in a number of cases. The tube can usually be removed in a comparatively few days.

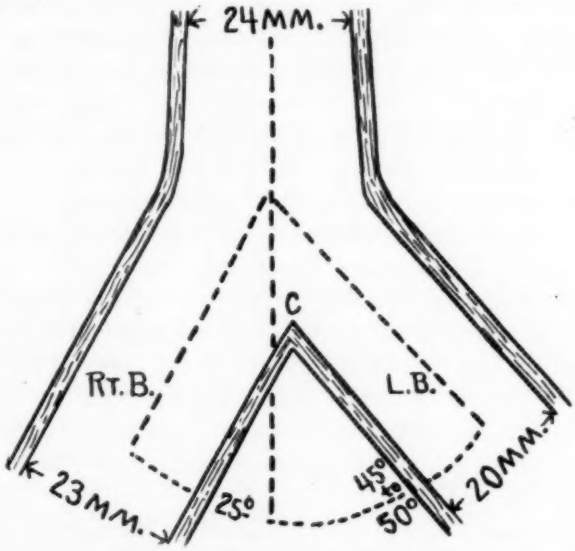

Fig. 5.—Diagram showing three anatomic reasons for the greater frequency of right sided lodgment of foreign bodies in the bronchi. The right bronchus (Rt. B.) is almost as wide (23 mm.) as the trachea (24 mm.); and it deviates from the long axis of the trachea much less than the left. The carina (C) is to the left of this axis. (After Sir St. Clair Thomson.)

CONCLUSIONS

1. It seems reasonable to believe that foreign bodies in the air and food passages in children are much more common than was formerly supposed. Statistics tend to show that about 66 per cent. of these cases of foreign bodies in the air passages occur in children.

2. The period of latency of symptoms which follows the violent dyspnea and choking attack, and later the gradual onset, and chronic character of the symptoms, might lead one to fail to suspect the presence of a foreign body.

3. Foreign bodies are certainly often overlooked, as a study of the histories of many cases clearly shows.

4. The symptoms vary greatly. The peanut kernel immediately sets up a severe laryngitis, tracheitis and bronchitis. In the peanut cases, the older child may survive the acute symptoms, but almost surely will develop pneumonia.

5. Metal objects may remain in the lung for a very long while and do comparatively very little damage.

6. There are some foreign bodies that do not cast a shadow on the plate.

7. The location of a foreign body in the esophagus that does not cast a shadow on the plate may often be diagnosed by allowing the patient to swallow a bismuth filled capsule. The roentgen ray shows the location of the bismuth capsule held in position in the esophagus by the foreign body.

8. Do not urge the patient to cough with the hope of expelling the foreign body inhaled in the lungs.

9. Foreign bodies are very rarely coughed up.

10. The physical signs and symptoms vary according to the composition, form, shape and size of the foreign body.

11. One should suspect a foreign body if the following conditions are present: an unexplained leukocytosis, localized symptoms in one lung that do not clear up under treatment, no tubercle bacilli in the sputum, and a gradual failure in weight and strength.

12. There are no contraindications to bronchoscopy except, perhaps, extreme weakness in the patient, when time should be given the individual to rally partially.

13. Bronchoscopy should be performed as soon as possible after the entrance of the foreign body.

14. Children do not require the administration of an anesthetic for the performing of a bronchoscopy.

15. The necessity of taking a roentgenogram in every case with a history of swallowing or inhaling a foreign body, cannot be emphasized too strongly.

16. The asthmatoid wheeze, when present, is a sign of considerable importance.

AURICULOVENTRICULAR HEART BLOCK IN CHILDREN

WITH REPORT OF A CASE *

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Cases of auriculoventricular dissociation in children that have been studied and described are summarized in the accompanying table. The case to be described in the present paper is also included. It would seem from the relatively small number of cases that a search of the literature has revealed that this condition is either much less common or is more frequently overlooked in children than in adults.

The diagnosis in Cases 1, 2, 3, 4, 11 and 16 was made by the ordinary methods of physical examination or inferred from the history of slow pulse and syncopal attacks. It must be accepted, therefore, with a certain amount of reservation. In the other cases, the condition was definitely constituted by either jugular pulse tracings or electrocardiographic records.

Probably the first case reported was that by Schuster, which the author states was one of bradycardia of unknown nature following acute rheumatic fever. Attacks occurred which simulated the Stokes-Adams syndrome, but were not absolutely typical. The pulse rate was normal between attacks. There was associated mitral insufficiency and extensive cardiac enlargement.

It will be noted that a large proportion of the cases reported occurred during severe and usually fatal cases of diphtheria. This group includes six of the twenty cases that we have been able to find in the literature. Five of these cases had a fatal termination. The predilection of the diphtheria toxins for heart muscle is well recognized clinically, and disturbances of the conduction system of the heart would probably be found more frequently if more carefully watched for in this disease. In addition to the cases summarized, two probable cases of heart block in eleven fatal cases of diphtheria studied by Rohmer[1] might be included. Because of some doubt as to the diagnosis and the absence of sufficient clinical data in regard to these cases, we have not included them in the summary. Further, in a series of fifteen fatal cases of diphtheria in which the hearts were subjected to careful pathologic study by Tanaka,[2] the case report on one indicated complete dissociation with the Stokes-Adams syndrome.

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1. Rohmer: Jahr. f. Kinderh. **76**:39, 1912.
2. Tanaka: Virchows Arch. f. path Anat. **207**:115, 1912.

The largest group of cases, nine in number, comprise those in which the cause of the disturbed conduction is ascribed to a congenital defect in the conductive system. The familial tendency in congenital cases has been emphasized by Marquio and by Fuller, Judson and Norris. Marquio's cases occurred in a family of eight children, five of whom manifested symptoms of Stokes-Adams' disease, beginning in all cases at 4 years of age and leading to the death of four of these patients before the ninth year. The fifth patient was still alive at 5 years of age. One of the three who showed no evidences of the disease died of typhoid fever at 6 years of age, one was 2 years old and thus below the age of manifestation of the disease in this family and one was 17 years of age and had shown no evidences of the disease. The father and mother were in good health with apparently normal hearts. The father and one sister of the case described by Fuller, Judson and Norris showed partial auriculoventricular dissociation and another daughter, although the heart was apparently normal at the time of examination, gave a history of previous attacks closely resembling the Stokes-Adams' syndrome.

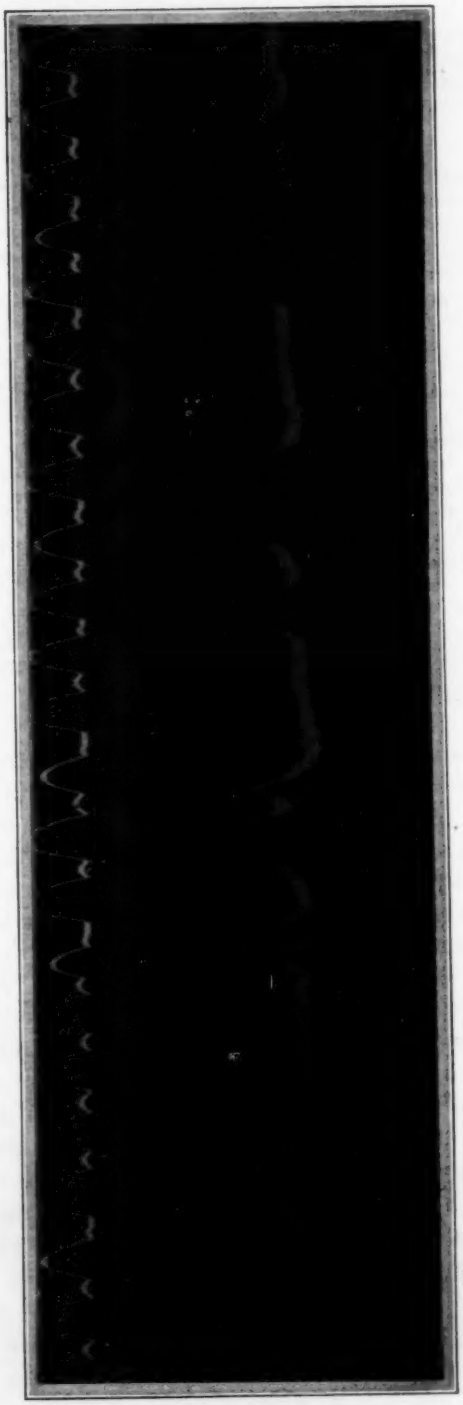
Of the remaining cases, one (Case 1) was associated with a severe mitral lesion resulting from acute articular rheumatism; one case (Case 12) was ascribed to the presence of a primary cardiac tumor believed to have its origin in the auriculoventricular node, and in one case (Case 17) the condition was discovered during a severe attack of whooping cough and bronchitis. In one case (Case 13) there was a history of measles four years and "inflammation of the heart valves" one year previous to the examination. Finally, Case 19 apparently developed during an acute febrile attack of undetermined nature.

The case forming the subject of the present report is one of partial auriculovenricular dissociation which has been under observation for more than two and a half years.

REPORT OF CASE

CASE.—A male child, aged 2 years, was referred by Dr. H. E. Purcell of Madison, Jan. 26, 1917, because of cardiac murmur and irregularity of rhythm. The family history is unimportant. The child was delivered by low forceps after normal pregnancy and at full term, and appeared normal up to the onset of the present trouble, although no cardiac examination was made previous to this time. The present illness began January 16 with an acute nasal and tonsillar infection, and examination by Dr. Purcell on the third day of the illness revealed the cardiac arrhythmia and murmur.

On examination the pulse was somewhat irregular, averaging about 75 per minute, and a soft systolic murmur was heard at the apex, transmitted to the midaxillary line, but not heard at the base. Electrocardiograms showed increase in auriculoventricular conduction and blocked auricular beats every third or fourth cycle. The temperature was normal. The tonsils were enlarged, particularly the left, which was also deeply congested. Other examinations were negative. There were no indications of congenital heart disease. Examinations on successive days showed an increase in the degree of heart block



2:1 Auriculoventricular heart block.

and an increase in the loudness and transmission of the murmur. Electrocardiograms made January 30 revealed a 2:1 auriculoventricular block, with a ventricular rate of 62. Roentgenograms showed enlargement of both ventricles and of the left auricle, with a total area of 57 sq. cm., corresponding, according to Bardeen's tables, to the normal for 8 years of age and a body weight of 48 pounds. There was considerable cyanosis of the head and of the body.

In view of the increase in the degree of block, change in character and transmission of the murmur and cardiac enlargement, it was believed that the pathologic process was definitely progressing. Because of the probable association of the tonsillar infection with the condition, tonsillectomy was performed February 2 under ether anesthesia. Cultures from the tonsils revealed *Staphylococcus aureus*. Following the operation, the condition improved steadily. February 7, the cyanosis had disappeared completely. The heart block was less, only every third or fourth auricular impulse failing to reach the ventricle. The pulse rate was 77. February 14, electrocardiograms showed a 3:2 heart block with a ventricular rate of 82. The extent of the block still further decreased and June 2, only every third or fourth beat was blocked. The murmur became soft and distant, and its area of propagation was reduced, being confined to the apex and its immediate vicinity. The cardiac size was reduced to 50½ sq. cm.

The child has been in good health since this time, has developed normally and manifests normal activity. July 20, 1919, a soft systolic murmur could be heard at and in the immediate neighborhood of the apex. The heart was moderately enlarged; the rate of beat was about 57 per minute and force and rhythm were regular. Electrocardiograms showed a 2:1 auriculoventricular heart block, as is shown in the accompanying illustration.

Administration of atropin in full doses increases slightly the rate of both auricles and ventricles but does not affect the degree of block. The depression of conductivity in the bundle is, therefore, not due to hypervagotonus.

DISCUSSION

The electrocardiograms at the first examination showed the mildest type of auriculoventricular heart block. Auriculoventricular conduction following a blocked beat was normal (from 0.14 to 0.16 second). There was then an increase in conduction time in the next two or three cycles, 0.30 to 0.32 second, and following this, conduction fails for one cycle. This condition passed a few days later into a condition of 2:1 block in which, however, occasionally two auricular beats in succession were conducted through to the ventricles. When the latter occurred, the period of auriculoventricular conduction in the second cycle was greatly prolonged. This increase in the extent of the block was associated with increase in the extent and distribution of the murmur, and evidences of deficient circulation (cyanosis) due to the slow ventricular rate. Following tonsillectomy, the conductivity of the auriculoventricular bundle improved, and with it the general condition of the patient. There was not, however, complete restoration of normal conductivity. Subsequent to this, the block again increased, without, however, any apparent interference with health and development. July 20, 1919, it was a 2:1 block with the occasional propagation of two successive auricular beats. When the latter occurs, the period of conduction in the second cycle is greatly prolonged. The character and

Summary of Cases Recorded in the Literature

Case	Date	Author	Reference	Age	Sex	Disease	Nature of Block	Character	Duration	Ventricular Rate	Termination	Stokes-Adams Syndrome
1	1806	Schuster	Deutsch. med. Wchnschr., **22**:484	4 yrs.	M	Rheumatism	?	Recurrent	3 mos.	35	.....	Present?
2	1901	Marquio	Arch. de med. d. enf., **4**:467	8 yrs.	M	Congenital	Complete?	Continuous	4 yrs.	50–70	Death	Present
3	1901	Marquio	.....	5 yrs.	M	Congenital	Complete?	Continuous	18 mos.	50–70	.....	Present
4	1903	v. Stark	Monatschr. f. Kinderh., **2**:11	5 yrs.	M	Congenital?	Complete?	Continuous?	1 yr.	28–44	Death	Present
5	1908	Dunn	J. A. M. A., **50**:1985	11 yrs.	M	Diphtheria	?	Continuous?	3 days	18–40	Death	Present
6	1908	v. d. Heuvel	Proefschr. v. Groeningen	13 yrs.	F	Congenital	Complete	Recurrent	11 yrs.	34	.....	Present
7	1910	Fulton, Judson and Norris	Am. J. M. Sc., **140**:339	2 yrs.	M	Congenital	Complete	Continuous	.....	40–50	.....	Absent
8	1910	Magnus-Alsleben	Ztschr. f. klin. Med., **69**:82	8 yrs.	M	Diphtheria	Complete	Recurrent	5 days	29–38	Death	Present?
9	1910	Fleming and Kennedy	Heart, **2**:77	10 yrs.	F	Diphtheria	Complete	Continuous	3 days	46–54	Death	Absent
10	1911	Price and Mackenzie	Heart, **3**:233	9 yrs.	F	Diphtheria	Complete	Continuous	5 days	34–48	Death	Absent
11	1911	Heilbecker	Ztschr. f. Path., **8**:319	14 yrs.	M	Diphtheria	Complete	Recurrent	3 days	5	Recovery	Present
12	1911	Armstrong and Monckeberg	Deutsch. Arch. f. klin. Med., **102**:143	5 yrs.	M	Cardiac tumor	Complete	Recurrent	18 mos.	28	Death	Present
13	1914	Hecht	Wien. med. Wchnschr., **64**:178	9 yrs.	M	?	Complete	Continuous	3 yrs.?	40	.....	Present
14	1914	Hecht	.....	3 yrs.	M	Diphtheria	Partial, later complete	Continuous	6 mos.	30–60	Recovery	Present
15	1915	d'Espine	Bull. de l'Acad. de méd., **74**:372	8 yrs.	M	Congenital	Complete	Continuous	1 mo.	20–32	.....	Present
16	1915	Zahorsky	Interstate M. J., **22**:57	15 mos.	F	Congenital	Complete	Continuous	14 mos.	50	.....	Present
17	1915	Whipham	Brit. J. Child. Dis., **12**:321	18 mos.	F	Bronchitis	Partial	Continuous	8 mos.	56–64	.....	Absent
18	1915	Whipham	.....	12 yrs.	F	Congenital?	Complete	Continuous	6 yrs.?	40–64	.....	Absent
19	1916	Frank and Polak	Nederlandsch. Tijdschr. v. Geneesk., **52**:2172	2 yrs.	F	Pyogenic	Complete	Continuous	.....	42–48	.....	Absent
20	1918	Bass	J. A. M. A., **70**:287 (Feb. 2)	15 yrs.	M	Congenital	Complete	Continuous	.....	37	.....	Absent
21	1919	Eyster and Middleton	.....	2 yrs.	M	Pyogenic?	Partial	Continuous	2½ yrs.	58	.....	Absent

distribution of the systolic murmur, and the size and shape of the heart, indicate the presence of a well compensated mitral lesion.

It seems to us that one of two possibe explanations can be given to the present case. The first is that of a congenital heart lesion of unknown nature, complicated by a lesion or imperfect development of the His bundle. Opposed to this is the probable absence of a murmur until the onset of the acute infection at 2 years of age, although careful physical examination had not been made previously, the atypical character of the shape of the cardiac outline, and the absence of any of the usual signs or symptoms of congenital heart disease. The second possible explanation is a myocardial focus of infection, possibly associated in its development with the attack of tonsillitis. The rapid increase in the extent of the block at one time, and the improvement in auriculoventricular conduction and in general symptoms subsequent to tonsillectomy, would seem to be in favor of this view of the nature of the condition. The accompanying decrease in the degree of cardiac enlargement, and the apparent absence of left auricular dilatation at present, point to the same conclusion.

SUMMARY

A case is described of partial auriculoventricular dissociation developing in a child, aged 2 years, apparently associated with an acute nasal and throat infection. This child has been under observation for more than two years. At present the cardiac condition is that of a well compensated mitral lesion, associated with a 2:1 auriculoventricular block, with a ventricular rate between 50 and 60. The child has developed normally, and is at present in apparent good health and is normally active.

A search of the literature has revealed twenty reported cases of heart block in children. Nearly all of these were definitely or probably of congenital origin or occurred during the course of severe and usually fatal diphtheria. The present case is regarded of particular interest in reference to its probable origin and the relatively mild clinical disturbance produced by the condition.

ABSCESS OF THE LUNGS IN INFANTS AND CHILDREN

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During the past few years there have come under our observation fifteen cases of abscess of the lungs in young individuals. They are included in a series of 100 cases occurring at all ages which were previously recorded by one of us (H. W.). However, because of their special interest to pediatricians they are, perhaps, worthy of separate consideration.

ETIOLOGY

Three of these cases followed aspiration of a foreign body; five cases were subsequent to operation for tonsillectomy and seven cases may be attributed to pneumonia or other inflammatory lung conditions.

PATHOLOGY

The cases following tonsillectomy easily lend themselves to a study of the manner in which these abscesses may be produced. It is probable that during general anesthesia the purulent plugs which are expressed from the tonsillar crypts by the grasping forceps are aspirated into one of the smaller bronchi. The obstructed bronchus is further sealed by aspirated blood, which, after it has clotted, creates favorable conditions for the development of whatever anaerobic organisms may be present in the tonsillar plug.

There results a local inflammatory process, a pneumonitis, within which will always be found one or many bronchi with necrotic and dilated walls. Subsequently, the usual signs of gangrene of the lung — cough with fetid expectoration—are added.

It is difficult to determine the rôle played by the various organisms which are found in these cases. Although it is possible that streptococci and other aerobic organisms may be partly or entirely responsible for the initial pneumonitis, there is good reason to believe that from its inception, abscess or gangrene of the lung may be regarded as an anaerobic lung infection. One of the grounds for this belief is the uniform incubation period not only in the posttonsillectomy cases, but also in all other cases in which aspiration of septic material into the bronchi occurs. Thus, in thirty-five cases of aspiration abscess or gangrene, the onset of fetid expectoration invariably occurred on the thirteenth or the fourteenth day after operation or aspiration.

Evidently, general anesthesia, which was resorted to in all of our cases, stands in close relation to the production of these abscesses.

It would seem that considerations of safety would prompt the invariable use of a suction pump whenever the operation is performed under general anesthesia, as almost without exception it has proved an effectual safeguard against aspiration.

The so-called postpneumonic cases may be separated into two groups. In the first group, the patient apparently contracts a pneumonia, which runs an acute course. On the thirteenth or fourteenth day, however, just as in the cases following aspiration, the classical symptoms and signs develop. The definite incubation period and the frequency of involvement of the upper lobes, make it plausible that in these cases the mechanism is the same as in the aspiration cases. In the second group of postpneumonic abscesses, a bronchopneumonia, instead of resolving, persists, and after varying periods of time, gives rise to a chronic indurative pneumonia with multiple bronchiectases which may or may not become gangrenous. Mention should finally be made of cases of pleuropneumonic bronchiectases or abscess which may result from empyemas.

The location of the inflammatory process in the lungs varies with the etiology. In the postoperative abscesses and in the aspiration type of postpneumonic abscess, the disease is usually situated in the upper lobes. On the other hand, abscesses resulting from the aspiration of foreign bodies and the chronic bronchopneumonic type of bronchiectasis usually localize in the lower lobes. It is not improbable that the recumbent position during operation has some bearing on the upper lobe localization of these abscesses.

CLINICAL COURSE

The symptoms of acute lung abscess, taking the posttonsillectomy cases as a type, are as follows: Immediately, or several days after the operation, a distressing persistent cough develops. This is especially harassing at night, but it may also be present throughout the day. With the cough there is usually a rise of temperature. The temperature is fairly constant during the first weeks, with minor fluctuations. Later it may become intermittent, and there may be entire absence of temperature elevation for days. The physical signs of lung involvement are usually slight or absent during the early stages. Later, they may become definite, and the evidences of a cavity may be made out. This is in contradistinction to the adult cases in which the physical signs, even of an extensive process and a large cavity, are usually indefinite.

On the thirteenth or fourteenth day, signs of gangrene are noted, including fetid breath, putrid sputum and hemoptysis. The sputum is then profuse. Club fingers appear very early and disappear after the abscess has healed, sometimes earlier. During the course of the dis-

ease, complications, such as perforation of the abscess into the pleura with a resulting empyema or pyopneumothorax, severe hemoptysis or cerebral abscess, may make their appearance.

The further course of cases of abscess of the lung depends on the etiology. A survey of the outcome of more than thirty postoperative abscesses in children and adults shows that in about one third of the cases recovery occurs spontaneously. This always takes place within about two months after the onset. An abscess which lasts longer than this is apparently not susceptible of spontaneous cure. These cases go on with periods of improvement and of apparent cure. However, sooner or later other abscess cavities form, extensive induration of the lungs develops, other lobes become secondarily infected and finally, any of the complications mentioned may supervene.

ANALYSIS OF FIFTEEN CASES OF ABSCESS OF THE LUNG

Etiology	Duration	Procedure	Result
Posttonsillectomy	6½ mos.	Operation	Death; hemorrhage
Posttonsillectomy	10 mos.	Operation	Cure
Posttonsillectomy	7 wks.	Palliative	Cure
Posttonsillectomy	.....	Operation	Death; shock
Posttonsillectomy	1 yr. 6 mos.	Palliative	Death; hemorrhage
Posttonsillectomy	5 wks.	Palliative	Death; gangrene of lung
Foreign body	8 yrs.	Palliative	Death; brain abscess
Foreign body	1 yr.	Operation	Cure
Foreign body	5 mos.	Bronchoscopy	Cure
Postpneumonia	8 mos.	Operation	Cure
Postpneumonia	10 mos.	Operation	Cure
Postpneumonia	6 yrs.	Palliative	Invalidism
Postpneumonia	6 yrs.	Palliative	Invalidism
Postpneumonia	8 yrs. plus	Palliative	Invalidism
Postpneumonia	6 wks.	Palliative	Cure; hemiplegia

PROGNOSIS

The prognosis is bad in the postpneumonic cases. In few of the cases is recovery spontaneous. Some of them pursue a course similar to that of the postonsillectomy cases. In the others, usually those following influenza or bronchopneumonia, minor symptoms may be manifested for a long time. The lung induration may lead to multiple, thin walled, cylindric bronchiectases which secrete enormous amounts of purulent sputum. In such cases, in which the disease does not involve the lung parenchyma, the patient may be well for years and suffer no more discomfort than is entailed by the periodic emptying of the numerous bronchiectases. On the other hand, the supervention of anaerobic infection may at any time convert these relatively benign cases into the more severe forms. They may continue over a period of many years.

The roentgen-ray examination has been of great value, not only for diagnosis, but also for a careful and exact oversight of the progress of these cases. Thus, in view of the frequent remission in the symp-

toms of acute abscess, great caution must be observed in making any statement in regard to cure. In cases of apparent cure the roentgen-ray examination has frequently demonstrated the persistence of a small pneumonic area, which has later been the nidus of a reinfection of the lung. Of course, the exact localization of the process is of the greatest value to the surgeon who contemplates any operative procedure.

TREATMENT

Certain indications for the treatment of these cases follow logically from what has been said. In at least one third of the postoperative cases recovery occurs spontaneously up to two months, and therefore it is evident that during this period, all treatment should be palliative and will differ little from that employed in any case of pneumonia. In cases which have persisted beyond this time the question of operative procedure will arise. The extremely distressing cough and nauseating odor of the expectoration make the patients not only a burden to themselves, but to those around them, and any operation that holds out even a small hope of success is eagerly welcomed. In young persons, with their great resistance to operative shock, the very serious operation of lobectomy, in selected cases, has yielded brilliant results, and it is hoped, that with further improvement in the technic of thoracic surgery, this operation may be still further freed from its dangers. Incision and drainage of the abscess offers less hope for permanent cure, although the operation in cases may result in considerable amelioration of the symptoms.

Finally, it may be emphasized that bronchoscopy should be performed in every case of lung abscess for the detection of a foreign body. In children especially a history of the aspiration of a foreign body may not be obtained, and the roentgen-ray examination may not disclose it. Thus, on more than one occasion a localized empyema has been traced to a ruptured lung abscess at the basis of which a foreign body was found by the bronchoscopist. Early removal of such a foreign body may lead to the prompt resolution of a lung abscess if it is not of too long standing.

THE ELIMINATION OF ACETONE BODIES DURING INFECTIOUS FEVERS*

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It has been established by numerous qualitative studies that acetone bodies are found in the urine of patients with infectious diseases. But few quantitative studies have been made in regard to this point, and these by early and rather imperfect analytical methods. As a part of a general study of acetonuria, we have collected some data in regard to the degree of acetonuria occurring in some of the infectious diseases of childhood, using the method of Schaffer for determining the quantity of acetone bodies.

Observations were made on forty-one children with scarlet fever, diphtheria, measles and pneumonia. In the majority of the observations, the collection of urine was started immediately on the admission of the patient to the hospital or after the onset of the disease. In a number, however, the infectious disease developed or was contracted during the presence of the patient in the hospital for some other condition, and complete curves from the very onset were obtained in these cases. The acetone, diacetic acid and betahydroxy-butyric acid were determined in terms of acetone.

In a previous paper[1] we showed that the healthy child eliminates on an average from 50 to 80 mg. of acetone bodies daily and the limits were from 20 to 100 mg. Accepting these figures, we find that only twenty-four of the forty-one children observed showed an increased elimination for some twenty-four hour period during the height of the disease, and in a number of them the excess was very slight. For example: D. H., scarlet fever. First day, 0.031 mg.; second day, 0.126 mg.; third day, 0.029 mg. The highest value obtained was 1.32 gm. eliminated in twenty-four hours by a patient with scarlet fever.

DEGREE OF FEVER AND SEVERITY OF INFECTION

The question next arises as to whether or not the acetonuria bears a relation to the severity of the infection. The degree of febrile reaction and the degree of prostration are the signs by which we are

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1. Veeder and Johnston: Am. J. Dis. Child. **13**:291 (April) 1916.

accustomed to judge the severity of infection — at least in the acute stage. Increased elimination, with a slight elevation of temperature, was uncommon, but, on the other hand, the number of instances in which an increased output of acetone bodies accompanied an elevation of temperature was about equal to the number of cases in which patients with an elevation of temperature showed no increased elimination of ketones. There is no measure of the degree of prostration, except the judgment of clinical experience. We were unable to link up in any way the severity of the infection, as indicated by the degree of prostration, with the amount of acetone bodies eliminated in the urine. In Table 1 a few cases are cited to show these conflicting points.

TABLE 1.—Analysis of Degree of Fever and Severity of Infection

Name, Age, Condition	Period	Temperature	Acetone and Diacetic Acid	B-Hydroxybutyric Acid	Food
I. B., 8 yrs.	1st day	104-106	0.120	0.059	Milk, 200 c.c.; vomited once
Lobar pneumonia	2d day	102-105	0.245	0.124	Milk, 800 c.c. and toast
Delirious	3d day	105- 98	0.265	0.188	Milk, 200 c.c. and toast
M. L., 9 yrs.	1st day	101-103	0.330	0.333	"Took food well"
Scarlet fever	2d day	101-103	0.140	0.016	Milk, 600 c.c.
Prostrated	3d day	101-103	0.818	0.238	Milk, 560 c.c.
R. F., 15 mos.	1st day	101-103	0.010	0.013	Took all feedings well
Bronchopneumonia	2d day	102-104	0.016	0.013	throughout period; no
Extreme prostration	3d day	102-104	0.042	0.035	vomiting
	4th day	102-103	0.017	0.023	
	5th day	100-101	0.037	0.038	
M. M., 8 yrs.	1st day	103-104	0.098	0.108	Milk, 300 c.c.; vomited
Lobar pneumonia	2d day	103-104	0.185	0.197	Milk, 175 c.c.
Prostrated					
E. L., 5 yrs.	1st day	100	0.306	0.967	Milk, 900 c.c. and bread; no vomiting
Scarlet fever					
Not prostrated	2d day	101-100	0.135	0.082	Milk, bread; ate well
	3d day	100-101	0.140	0.065	Milk, 450 c.c.
S. J., 6 yrs.	1st day	100	0.355	0.688	"Took a little milk"
J. H., 14 mos.					
Lobar pneumonia	1st day	103-105	0.030	0.014	Took food; no vomiting
Extreme prostration (12 hrs. to death)	2d day	103-105	0.008	0.007	Took no food

INANITION

In a previous paper[2] we have shown that, as a rule, children show a very moderate increase in the production of acetone bodies as the result of inanition for periods of from twenty-four to forty-eight hours. The cases of infectious disease showing acetonuria usually took food, and, further, the increased acetonuria was in most of the cases out of all proportion to the inanition factor. Although it may not be disregarded entirely, it is by no means possible to explain the acetonuria in the infectious conditions on the basis of inanition.

2. Veeder and Johnston: Am. J. Dis. Child. **13**:89, (Jan.) 1917.

UNDETERMINED FACTORS

We were fortunate in obtaining figures on a number of children with cross or secondary infections. That is, determinations were made at the time the patient was admitted with one infection and again later during a second infection developing while the child was in the hospital. Both the absolute number of patients developing acetonuria, and the amount of increased elimination in those showing this acetonuria during the second period was decreased. This is strikingly shown in the cases of C. M. and E. D. (Table 2).

It will be noted that in both these cases the high point occurred during the twenty-four hours immediately after admission. This was true of nearly all of the patients showing acetonuria. In both of these cases the acetone bodies in the second periods did not exceed normal figures. The simplest explanation for this is to assume some psychic factor by which the excitement of moving the patient, bringing him to the hospital, and the new surroundings, produce a reaction not found when the infection develops amid quiet surroundings to which the child is accustomed. It is impossible, of course, to demonstrate the proof of this explanation.

TABLE 2.—SHOWING ELIMINATION OF ACETONE DURING FIRST AND SECOND PERIODS

	Period	Temperature	Acetone	B.
C. M.: Scarlet fever				
12/13-13/16	I (2d day of disease)	102-104	0.380	0.650
	II	102-104	0.331	0.488
	III	102-104	0.205	0.118
Measles				
2/ 1-2/ 2	I	101-103	0.033	0.024
1/12-1/15	II	103-105	0.020	0.006
E. D.: Lobar pneumonia	I (4th day of disease)	105	0.140	0.188
	II	105- 99	0 040	0.065
	III	98	0.027	0.056
Scarlet fever	I	103-104	0.010	0 030
	II	100-103	0.012	0.035
1/15-1/18	III	100-104	0.005	0.024

Our findings do show definitely, however, that while an increased elimination of acetone bodies may occur in the infectious diseases, this does not always take place; that in the same patient it may occur during one infection and not during a second; that it is not dependent on the severity of the infection or the degree of temperature; and lastly that the decreased intake of food, so consistently a part of an infectious process, does not explain the causation.

CLINICAL DEPARTMENT

A REPORT OF TWO CASES OF CONGENITAL STRICTURE OF THE ESOPHAGUS*

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These cases are reported partly because of the rarity of the condition and partly because the symptoms, although very characteristic, are usually misinterpreted and attributed to some other disease.

REPORT OF CASES

CASE 1 (No. 19217).—A boy was admitted to the Children's Hospital when 6 years old. His parents and seven other children were well. One child had died in the Children's Hospital some years before after an operation for esophageal stricture. Further details regarding this case will be given later. The mother had had two miscarriages.

The patient was born at full term after a normal labor and was thought to have been normal at birth. He was breast fed and seemed well in every way until the attempt was made to feed him when he was 1 year old. It was then found that while he was able to take liquids he could not take solid food without vomiting. On this account he was nursed until he was 2½ years old. Since then, he had been able to take liquids and soft solids, but no solid food. Solid food was vomited immediately after it was swallowed. In addition he had spells of vomiting lasting two or three days every two weeks or oftener. These attacks were apparently not dependent on the character of the food. The vomiting had never been projectile. It always occurred immediately after taking food. If he did not vomit then, he did not vomit at all. The vomitus had never contained blood or bile, but was made up solely of food. He never vomited at night. His appetite was poor; his bowels were regular. He had developed normally, creeping at 8 months, walking at 9 months, saying single words at 9 months, and making sentences at 17 months. He had been seen by many physicians, all of whom had made the diagnosis of indigestion and had prescribed various remedies and diets.

He was poorly developed and fairly nourished, weighing 32¾ pounds, and measuring 40 inches in length, while the average for his age is 45 pounds and 42 inches, respectively. He was somewhat pale. He had the characteristic adenoid facies. His teeth were bad, and his lower jaw protruded. D'Espine's sign was negative. There was no dulness under the manubrium or over the spinous processes, and air entered both lungs alike. The rest of the physical examination showed nothing abnormal. The hemoglobin was 55 per cent.; the leukocyte count, 25,000. The tuberculin test was negative, and the urine was normal.

His story in the hospital was the same as it had been outside. He was unable to take solid food without vomiting immediately. At times he took liquids and soft solids without trouble; at other times he vomited them also at once.

Roentgenograms taken after a barium meal showed, according to the report of Dr. Percy Brown, "distinct enlargement of the esophagus at about the level of the sternoclavicular area. The course of the esophagus is likewise well seen below the point of stenosis. From the persistency of the shadow, its cause is believed to be organic rather than spastic." (Figs. 1 and 2.)

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*From the medical service of the Children's Hospital, Boston.

Esophagoscopy under ether by Drs. Herman and Pratt showed an obstruction six inches from the incisor teeth which was readily dilated. There was also some constriction at seven inches and onward, more suggestive of congenital narrowing than inflammatory stricture. The stricture was dilated up to a No. 23 bougie, which passed into the stomach with only slight bleeding.

The boy returned a month later for further dilatation. He had gained a pound in weight and had not vomited until about a week before he came back. Dilatation will, of course, have to be continued at intervals.

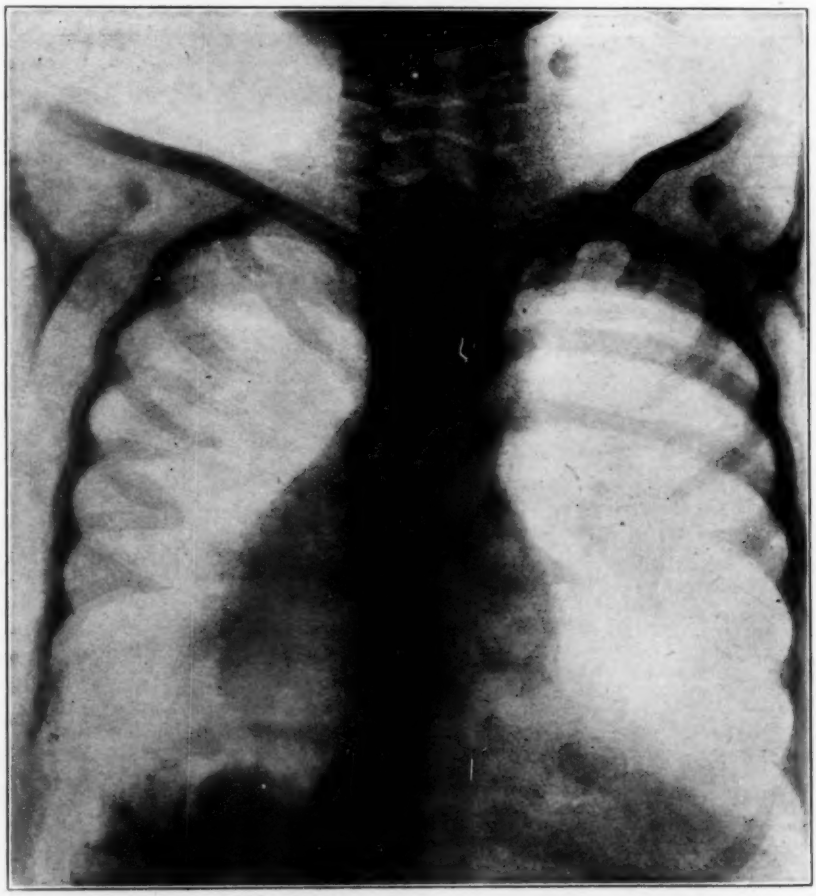

Fig. 1.—Case 1. Fusiform dilatation of the esophagus at sternoclavicular junction and narrowing below this point. Anteroposterior view.

CASE 2 (No. 5028).—This patient was brought to the hospital seven years ago when he was 25 months old. He is a brother of the other patient. His case was reported by Dr. T. M. Rotch.[1] In brief, he had vomited from four to six times a day ever since birth. The vomiting had always occurred during feeding and had never been forcible. It made no difference whether the food was breast milk, milk or Mellin's food. He often retained several feedings and then vomited two or three in succession.

1. Am. J. Dis. Child. **6**:1 (July) 1913.

His general physical examination was negative, except that he was thin and pale. A catheter was stopped 15.5 cm. from the incisor teeth. The roentgenogram after a bismuth meal showed a narrowing of the lower one-third of the esophagus with dilatation above. Esophagoscopy by Dr. D. Crosby Greene showed a stricture of the esophagus 17.5 cm. from the incisor teeth. This was approached by a funnel shaped narrowing, after which the esophagus was slightly dilated. The stricture was very small, looking about the size of the lead of a lead pencil. It was deemed inadvisable to attempt to dilate the stricture and a gastrotomy was done. He died a week later.

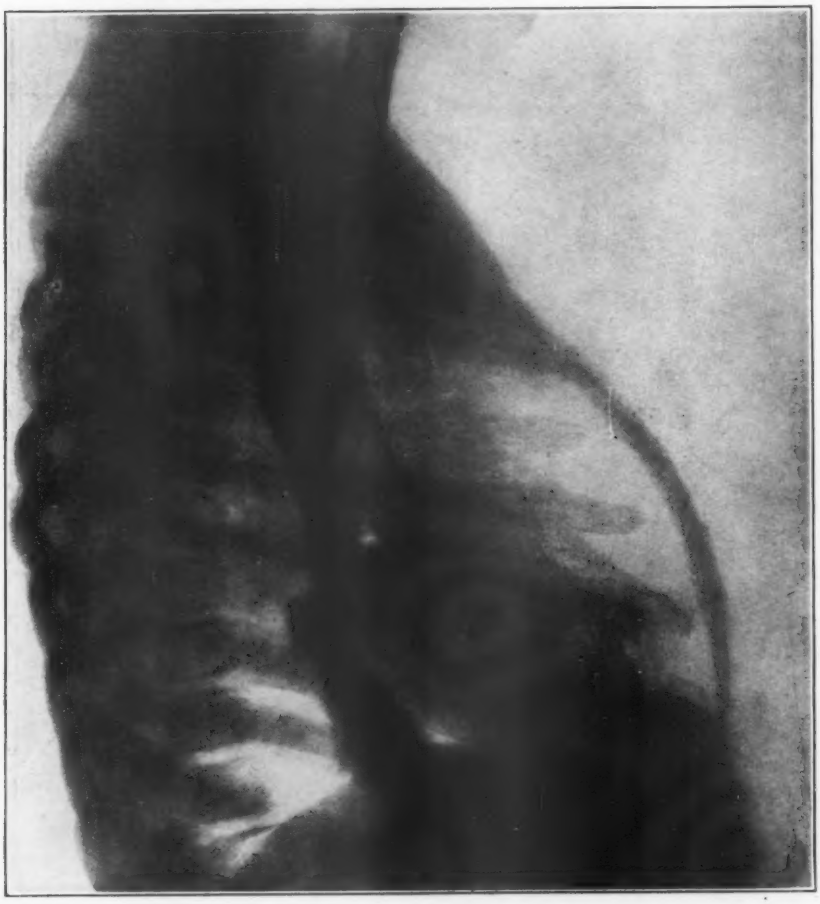

Fig. 2.—Case 1. Lateral view showing distinctly the enlargement and the constriction below it.

The occurrence of a deformity of this sort in two members of the same family must be most unusual.

Case 3 (No. 14082).—A girl was admitted to the Children's Hospital when 3 years old. Her parents were well. She was an only child. She was born at full term after a forceps delivery. She was normal at birth and weighed 7½ pounds.

She began to vomit soon after birth. Nursing was given up on this account when she was 2 weeks old. The vomiting, however, continued. It occurred immediately after feeding, never before or at some time after. There were long intervals during which she did not vomit milk or any liquid food. She never failed, however, to vomit immediately whatever solid food was given, even if it was only a small amount. She had been treated by many doctors for the vomiting, but the diets which they had prescribed and the medicines which they had given had never had any effect on the vomiting.

She had done fairly well, however, until six days before she entered the hospital when she began to vomit everything given. She had been unable to retain even water. The bowels had not moved for three or four days, in spite of suppositories. The urine had been scanty and she had probably had a little fever. She was sent into the hospital with the diagnosis of "acidosis," and a question of cardiospasm. The physical examination showed nothing abnormal. The hemoglobin was 76 per cent.; the leukocyte count, 23,600. The tuberculin and Wassermann tests were negative. She weighed 25 pounds.

She was able to swallow without difficulty, but, after taking about ½ ounce, she complained of being sick and then regurgitated without difficulty all the fluid taken. The fluid did not seem to come from the stomach and there was no retching. It was mixed with mucus, but the odor was not acid. The stomach tube was stopped seven inches from the incisor teeth.

Esophagoscopy under ether by Dr. Greene showed that the esophagus was normal to the point of stricture, seven and a half inches from the incisor teeth where there was a small circular opening showing no cicatrix and admitting a No. 7 French bougie. There was no marked dilatation above the stricture. Bougies were passed up to No. 16. She has been dilated repeatedly and now, after eight months, has no symptoms.

PROGRESS IN PEDIATRICS

RECENT WORK IN ANATOMY, PHYSIOLOGY AND PATHOLOGY OF INFANCY AND CHILDHOOD

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ANATOMY

Significance of Striations in Muscle.—Lewis,[1] in some studies on the cells of heart muscle from very young chick embryos, arrives at some conclusions that are of far reaching application. She finds it possible to study with satisfaction the living cells of heart muscle from embryos of ten myotomes. In these cells she finds that complete cross striations are present, and much earlier than has been supposed by other observers. Cross striations are present, but not fibrils. The cross striations are very thin bands on the surface of the cell. They extend across the cell and never appear as narrow threads or fibrils. Fixation of the cell causes the formation of the surface layer into fibrils on which the cross striations are drawn together into deeper bundles, and then become evident as sharply marked structures. In places where the pull on the surfaces of the cell is such that they are not coagulated into fibrils, the cross striations remain spread out as thin bands extending across the cell. No structure resembling the fibrils of the fixed preparation was present in the living heart muscle at any of the ages studied.

"The physiologists have endeavored without success to formulate a theory, based on the myofibrils, to account for the contraction of the muscle cells. It is not surprising that such a theory has not proved satisfactory, at least in regard to the heart muscle, since the structure on which it was based is not a part of the living heart muscle cell, but only of the dead cell. In other words, a cell containing the structure on which it was attempted to build the theory is not capable of undergoing contraction."

The ossification centers of the fetal pelvis receive further consideration from Rosmark.[2]

R. Taylor[3] contributes measurements obtained from 250 full term, newly born infants.

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1. Lewis, M. R.: The Development of Cross Striations in the Heart Muscle of the Chick Embryo, Bull. Johns Hopkins Hosp. **30**:176, 1919.
2. Rosmark, T.: The Ossification Centers of the Fetal Pelvis, Am. J. Obst. **78**:175, 1918.
3. Taylor, R.: Measurements of 250 Full Term New-Born Infants, Am. J. Dis. Child. **17**:353 (May) 1919.

Scammon[4] points out that while accumulated data regarding the growth of gastric capacity have been summarized from time to time in the studies of Pfaundler, Borie, Marfan and Figueira, it is now more than fifteen years since the last attempt at a synthesis of this material. Much valuable new material has accumulated. The material is presented by Scammon in an article that is replete with graphs and references to statistics.

The question of growth of gastric capacity has been approached from many angles, and it is noteworthy that graphic curves based on the data obtained by different methods are of the same general type. Curves of gastric capacity show an initial stage of rapid rise (during the first month of life) followed by a much longer stage in which the rise is slower and without important changes in rate. It is found possible to represent the curves by certain empiric formulas of a single type. In these formulas gastric capacity equals a numerical value plus a multiple of the age of the child expressed in weeks.

PHYSIOLOGY

Metabolism.—Olmstead, Barr and Du Bois[5] report further studies on the metabolism of growing boys. Two years ago Du Bois reported figures showing that the metabolism in boys 12 years of age is 25 per cent. higher than for an adult of the same height and weight. (As since expressed in other figures by Lusk: The basal metabolism of an average boy, 13 years of age, 4 feet 10 inches in height, and weighing 80 pounds, may be calculated as 1,525 calories a day. This is the same as that of a man, 25 years of age, weighing 126 pounds, and 5 feet 2 inches in height.) It is generally accepted that metabolism is relatively high during childhood, falling rapidly in adolescence, and then decreasing slowly during the rest of life. Du Bois has been fortunate in being able to reexamine certain of the boys whom he had previously studied, some of them now exhibiting signs of puberty. He finds a marked decrease in metabolism, a decrease of 13 per cent. per unit of body surface, for the seven boys studied. The metabolism is, however, still 11 per cent. above the average for adult men between 21 and 40 years of age, and about 4 per cent. above that of adult women.

The Stomach.—F. W. White[6] has studied the effect of stimuli from the lower bowel on the rate of emptying of the stomach. Studies were

4. Scammon, Richard E.: Some Graphs and Tables Illustrating the Growth of the Human Stomach, Am. J. Dis. Child. **17**:395 (June) 1919.

5. Olmstead, W. H., Barr, D. P., and Du Bois, E. F.: Clinical Calorimetry, Paper 27, Metabolism of Boys Twelve and Fourteen Years Old, Arch. Int. Med. **21**:621 (May) 1918.

6. White, F. W.: The Effect of Stimuli from the Lower Bowel on the Rate of Emptying of the Stomach, Am. J. M. Sc. **156**:184, 1918.

made on men, and, experimentally, on cats. The effects of various irritative lesions were studied. Delay in emptying a barium meal from the stomach was found to be the exception; strong stimuli were needed to slow the stomach. Marked delay in emptying the stomach is far more often the result of actual lesions about the pylorus than of reflexes from the bowel. The stomach emptied itself within the normal time in some cases of ileac stasis of two or more days' duration, and in most cases with good sized twelve hour residue in the ileum, also when the colon was distended with a large enema, also in most cases of chronic appendicitis and chronic inflammations and tumors of the colon. These gastric symptoms in intestinal cases are not, as a rule, the result of slow emptying of the stomach.

Sauer[7] has utilized thick farina feedings in twelve cases of pyloric stenosis in infants. In all but one case the effect of thick feedings was striking. In eleven of the twelve cases the vomiting soon stopped, although in most of the patients the peristaltic waves and the tumor (where palpable) persisted for weeks or months after vomiting ceased. The infants were seen when from 6 weeks to 2 months of age. The feeding was prepared as follows: One part of farina in seven parts of fluid (three parts of milk to four of water) was boiled in a covered double boiler for one hour. Such a food contains about 15 per cent. of cereal. Salt and sugar may be added. The infants are placed on the right side after feeding, and disturbed as little as possible. Small supplementary fluid feedings are added later.

The Intestine and Drugs.—A. Galvani[8] considers the administration of epinephrin. He remarks that the soft and elastic arteries in children, and the integrity of the cardiovascular and other systems, render epinephrin peculiarly effective in pediatric practice. He reviews the indications for it, the dosage, etc., as published by various writers. When especially prompt action is necessary, it may be administered subcutaneously, from 0.5 to 1 c.c.; but, except in urgent cases, he advocates administration by mouth, from 10 to 30 drops of the 1:1,000 solution. Its action is more protracted by mouth, and abrupt changes in the circulation are avoided. It is thought to stimulate the centers, possibly by way of the sympathetic system. The effect of the drug on the musculature of the intestines, Galvani notes, is yet undecided. Lesné, in 1912, reported that a given dose of epinephrin injected into the rectum of rabbits caused death, but not so rapidly as when a like dose was injected subcutaneously. The same dose introduced into the stomach or small intestine seemed entirely harmless.

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7. Sauer, L. W.: The Use of Thick Farina in the Treatment of Pyloric Stenosis, Arch. Pediat. **35**:385, 1918.
8. Galvani, A.: Rivista di Clinica Pediatrica **16**:225, 1918.

Binet[9] in a review of the literature, concludes that epinephrin has an undoubted modifying action on the vascularization, secretion and motor functioning of the digestive tract, and states that digestive disturbances from suprarenal insufficiency are known. Administered by mouth, epinephrin does not seem to display a toxic action; it does not appear to be acted on by pepsin or trypsin or passage through the intestinal capillaries. Administered by rectum, it is powerfully toxic. The writer ascribes this to the close anastomoses between the hemorrhoidal veins and the portal vein, the liver being apparently the barrier which arrests the ingested epinephrin.

Alvarez[10] has studied the action of drugs on different parts of the intestine. Other work convinced him that there are marked differences in the neuromuscular apparatus in different parts of the tract. He made use of segments of rabbit's intestine placed in warm aerated Locke's solution, and tested a great variety of drugs. He found that some drugs stimulated all parts equally; some stimulated in small doses but depressed in large doses; some depressed or stimulated after an opposite initial effect; some acted more powerfully in one part than another; a few actually stimulated one end and depressed the other. Epinephrin stimulated. Mercuric chlorid generally stimulated the colon less than the small intestine.

Dudodenum.—E. G. Grey[11] has shown that in the dog it is possible to remove the duodenum, transplanting the major pancreatic duct to the jejunum, and have the animal enjoy comparative good health for at least nine and one-half months, provided the pancreas and the liver continue actively to discharge their secretions into the intestinal tract. He thus refutes the conclusion of Stassoff that the duodenum is essential to life. The operation must be done in several stages, but every trace of the duodenal mucosa and approximately all of the muscular coats can be removed without embarrassing the vascular supply of the pancreas. Withdrawal of the alkaline juices from the duodenum, without removal of the tract, not infrequently leads to the formation of peptic ulcers.

Lactation.—E. L. Cornell[12] reports observations on the effect of placental tissue as a galactagogue. He says that 87 per cent. of the babies whose mothers had received placental tissue began to gain on the fourth and fifth days, against 69 per cent. whose mothers did not

9. Binet, L.: L'Action de l'Adrenaline sur le Tube Gastro-Intestinal, Presse méd. **26**:407, 1918.

10. Alvarez, W. C.: Differences in the Action of Drugs on Different Parts of the Bowel, J. Pharmacol. & Exper. Therap. **12**:171, 1918.

11. Grey, E. G.: Duodenectomy; Its Effect on the Life of an Animal. Transplantation of the Pancreatic Duct, Surg., Gynec. & Obst. **28**:36, 1919.

12. Cornell, E. L.: Placental Tissue as a Galactagogue, Surg., Gynec. & Obst. **27**:535, 1918.

receive this medication. Forty-four per cent. of the babies regained their birth weight before leaving the hospital, against 24 per cent. who did not.

Milk.—The presence of citric acid as a normal constituent of cow's milk was reported by Soxhlet in 1888. Sommer and Hart[13] have recently isolated and identified the compound anew. They find that it amounts to approximately 0.2 per cent. of the milk, or 2 per cent. of the milk solids. Citric acid is a recognized constituent of various "acid" fruit juices and vegetables, and to its presence in them some have ascribed their antiscorbutic properties. Sommer and Hart, therefore, studied the effect of heat on the citric acid content of milk. They find that it is not destroyed even by autoclaving the milk; nor are the citric acid salts of milk changed to an insoluble form by heating.

Bosworth and Giblin[14] report the preparation of a considerable quantity of pure casein from human milk. It is found to have the same nitrogen, phosphorus and sulphur content as casein prepared from cow's and goat's milk; it has the same degree of valency and gives the same series of salts with bases; it has the same molecular weight; it is acted on by rennin in the same manner; the paracasein produced by the action of rennin is similar to the paracasein produced by the action of rennin in the casein of cow's milk.

Van Slyke and Baker[15] describe a method of preparing pure casein from milk which is practically quantitative.

Barley.—Steenbock, Kent and Gross[16] present the results of experiments with barley in feeding growing animals. Barley alone does not meet the needs of the growing animal. It contains an abundance of water soluble vitamin, but is deficient in fat-soluble vitamin. The addition of butter-fat improves its growth promoting quality to some extent. Of single additions, a salt mixture, by satisfying the demands of the animal for elements in the group, calcium, chlorin and sodium, exerted the most beneficial effect. Where salts, protein and fat-soluble vitamin were all added, normal growth, reproduction and raising of young became possible.

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13. Sommer, H. H., and Hart, E. B.: The Effect of Heat on the Citric Acid Content of Milk. Isolation of Citric Acid from Milk, J. Biol. Chem. **35**:313, 1918.

14. Bosworth, A. W., and Giblin, L. A.: The Casein of Human Milk, J. Biol. Chem. **35**:115, 1918.

15. Van Slyke, L. L., and Baker, J. C.: The Preparation of Pure Casein, J. Biol. Chem. **35**:137, 1918.

16. Steenbock, H. E., Kent, H. E., and Gross, E. G.: The Dietary Qualities of Barley, J. Biol. Chem. **35**:61, 1918.

Calcium and Metabolism.—Sherman, Gillette and Pope,[17] and other co-workers, in a series of very careful and prolonged balance experiments, have shown that, in general, an average of 0.45 gm. (about 7 grains) of calcium per adult man per day represents approximately the minimum maintenance requirement for normal nutrition. This amount of calcium is contained in a pint or less of milk; there is less proportionately in meat or common cereals. Though calcium is the most abundant inorganic element in the body, the bones particularly forming a large store of it, nevertheless, as Osborne and Mendel[18] have recently emphasized anew, the supply of calcium cannot long be neglected with impunity in the diet of individuals that have a special need for it, as is true in growth and during gestation and milk production.

The mode of absorption of fats from the alimentary tract has long been a matter of controversy. The present view is that fats are chemically split in the gastro-intestinal tract into fatty acids and glycerols. The fatty acids react readily with bases to form soaps, and as such pass through the intestinal mucosa with varying degrees of facility. The soaps of sodium and potassium are somewhat soluble; those of calcium and magnesium tend to be insoluble. In the case of infants particularly the soaps of calcium are found in the stools. When present in large amounts, or accompanied by other symptoms, the appearance of soap (fatty curds) in the feces has been interpreted as evidence of faulty digestion of fats, or of "fat intolerance," and, as a rule, the amount of fat in the infant's food has been greatly reduced, and often with apparent improvement.

Recently the matter has been approached from a different angle by Bosworth, Bowditch and Gilbin.[19] They note that the calcium content of cow's milk is proportionately higher, compared with the other nutrients, than that of breast milk, a relation which is not altered by merely diluting the milk. This is true also of the phosphoric acid. Both the substances, when fed to infants in the proportions found in cow's milk or in modified cow's milk, are greatly in excess of the amounts supposedly required to supply the demands of the infant, and hence are eliminated either in the urine or in the feces. As the calcium metabolism of bottle fed infants, as measured by the calcium eliminated

17. Sherman, H. C., Gillette, L. H., and Pope, H. M.: Monthly Metabolism of Nitrogen, Phosphorus and Calcium in Healthy Women, J. Biol. Chem. **34**:373, 383, 1918.

18. Osborne, T. B., and Mendel, L. B.: The Inorganic Elements in Nutrition, ibid. **34**:131, 1918.

19. Bosworth, A. W., Bowditch, L. H., and Giblin, L. A.: Studies, etc., Calcium in Its Relation to the Absorption of Fatty Acids, Am. J. Dis. Child. **15**: 397 (December) 1918.

in the urine, is seldom greater, and often less, than found in breast fed infants, it appears that the excess of calcium must be eliminated in the feces, mostly as insoluble calcium phosphate and calcium soaps, or be ingested at the risk of entering the body fluids and tissues in organic combination and, perhaps, subsequently setting up a toxic condition.

Bosworth, Bowditch and Giblin have, therefore, devised a method by which they reconstruct cow's milk so as to remove much of the calcium. By the use of this reconstructed milk they think they have obtained favorable results in certain cases otherwise difficult to manage dietetically, and in cases classified as "fat intolerance" and "infantile atrophy." Reports of further work by these authors will be awaited with interest.

Meantime, Holt, Courtney and Fales[20] have attacked certain of these contentions. They deny that a high calcium intake necessarily causes a serious loss of fat in the feces. Great reduction of the calcium content of the food of infants may, on the other hand, be attended with considerable risk. These authors are of the opinion that it has yet to be demonstrated that infants fed on simple dilutions of cow's milk do not retain an adequate amount of fat, when the stools are formed, or semiformed and soapy. They warn that unless the harm caused by a fairly high calcium intake can be demonstrated quite definitely it would be safer to allow an excess of calcium in the intake rather than to run any risk of providing less calcium than is needed for the normal growth of the bones. The large calcium requirement of children with a tendency to rickets is mentioned; as is also the occurrence of tetany and rickets in children too long fed exclusively on breast milk.

Sato[21] reports experiments made with the same malt preparation on the same child to determine whether the beneficial effect on the calcium retention attributed to malt extracts is due to the extract itself or to the added alkali. He found that the addition of alkali to milk produced no favorable effect on the retention of calcium, but distinctly an unfavorable one. Malt extract alone appeared to act beneficially on calcium storage. Malt extract to which a considerable amount of alkali has been added seemed to act unfavorably. Thus, if malt soup has a favorable effect on calcium metabolism, it is not as the result of the alkali originally contained in it or added to it.

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20. Holt, L. E., Courtney, A. M., and Fales, H. L.: Is the Amount of Calcium Usually Given in Dilutions of Cow's Milk Injurious to Infants? Am. J. Dis. Child. **16**:52 (July) 1918.

21. Sato, A.: The Effect of Alkali and Malt Preparations on the Retention of Calcium in Infancy, Am. J. Dis. Child. **16**:293 (November) 1918.

Givens[22] and McClugage and Mendel[23] have studied two other phases of calcium metabolism experimentally (in dogs). The former, as the result of new experiments, is able to confirm his previous statement that ingested hydrochloric acid is without marked influence on the calcium and magnesium metabolism of the dog. There is an increased urinary elimination of calcium after ingestion of acid, but the absolute amount of this increase is not sufficient to affect to any noticeable degree the calcium balance.

McClugage and Mendel show that, in dogs at least, the calcium in milk is better utilized than that in calcium phosphate, and the calcium in spinach and carrots was poorly assimilated as compared with that in milk.

The outcome of analogous studies in infants, if such are possible, will be of interest. During the past two decades it has become increasingly the practice in infant nutrition to introduce suitable garden vegetables, such as carrots and spinach, into the infant's dietary, and this more especially where the onset of rickets was feared. Recently Courtney, Fales and Bartlett[24] presented some analyses showing the effect on these vegetables of the method of cooking employed. It is understood that they are comparatively rich in calcium, and their supposed value has been attributed in part to their effect on mineral metabolism.

Goldschmidt and Dayton[25] have studied the effect of sodium, calcium and magnesium salts on the absorption of sodium chlorid in the intestine. They find the colon is not characterized by a strictly one-sided permeability. Toward solution of sodium sulphate it behaves like a semipermeable membrane.

Further work by Bosworth and Bowditch[26] bears on the absorption of calcium. They report experiments to show that the ingestion of large amounts of calcium and its absorption as organic salts, if unaccompanied by the ingestion of sufficient phosphorus or chlorin to form soluble salts, may lead to an accumulation of calcium in the tissues, to be followed by a toxic condition and the elimination of

22. Givens, M. H.: Studies in Calcium and Magnesium Metabolism, etc., J. Biol. Chem. **35**:241, 1918.

23. McClugage, H. B., and Mendel, L. B.: Experiments on Utilization of Nitrogen, Calcium, and Magnesium, etc., ibid. **35**:353, 1918.

24. Courtney, A. M., Fales, H. L., and Bartlett, F. H.: Some Analyses of Vegetables Showing the Effect of the Method of Cooking, Am. J. Dis. Child. **14**:34 (July) 1917.

25. Goldschmidt, S., and Dayton, A. B.: The Mechanism of Absorption from the Intestine. I. Colon. One-Sided Permeability of Intestinal Wall to Chlorid, etc., Am. J. Physiol. **48**:419, 1919.

26. Bosworth, A. W., and Bowditch, H. I.: Infant Feeding. High Protein Feeding vs. High Calcium Absorption as a Cause of Increase of Body Temperature in Infants, Am. J. Dis. Child. **16**:279 (November) 1918.

calcium lactate in the urine. An accompanying high protein content in the food (a synthetic one) they thought responsible only to the extent that it might be a carrier of calcium in the form of calcium caseinate. Once in the body it was thought the calcium might act itself, or as a salt, possibly calcium oxalate.

PATHOLOGY

Of Schoolchildren.—In New Jersey, during the school year 1917-1918, 21,263 children were examined.[27] Of this number only 6,243 were reported to be normal, 15,020 were reported to be subnormal, or approximately 75 per cent. of the total number; 4 per cent. were found to be suffering from malnutrition; 5 per cent. from enlarged cervical glands; 15 per cent. with defective vision; 3.5 per cent. with defective nasal breathing; 5.5 per cent. with defective teeth; 8.5 per cent. with enlarged tonsils; 1.25 per cent. with impediment of speech.

As usual, the subnormal children were found to suffer chiefly from defects of vision, from unhealthy conditions of the adenoids and tonsils, and from faulty nutrition. It is obvious that wide latitude must be allowed to individual examiners as to what constitutes an abnormal condition of the adenoids and tonsils.

Malnutrition.—The Dunfermline scale groups children as I, II, III, IV, according to the state of their nutrition, and is based on their appearance only. Holt,[28] in a criticism of the figures given out by the New York Department of Health, writes that observations which do not take account of height and weight cannot be relied on as determining the child's nutrition. Figures based on inspection, and made without removing the clothing, cannot be accepted as accurately representing the amount of malnutrition existing among schoolchildren.

Anamolies.—Massini[29] reports the occurrence of an ectromelic monster. The roentgenograms of his case confirm the assumption that the so-called amputations are in reality malformations. The limbs show different malformations above the supposed amputation from constricting bands.

D. M. Greig[30] describes an instance of congenital tumor of the frontonasal region. The tumor was pyriform in shape, thick skinned, reddish, 1½ inches in length, and dependent from the region of the left frontonasal suture. It had a translucent appearance, its surface was glazed and smooth, and it was of a somewhat soft consistency.

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27. Public Health News (New Jersey), 1918; Abst. J. A. M. A. **72**:1289 (May 3) 1919.
28. Holt, L. Emmett: "Erratum," Arch. Pediat. **35**:512, 1918.
29. Massini, J. C. L.: Semana méd. **25**:365, 1918.
30. Greig, D. M.: Two Cases of Unusual Congenital Abnormalities, Edinburgh M. J. **11**:384, 1917.

It could not be diminished by pressure, and there was no evidence that it communicated with the interior of the cranium. A large double hare-lip and cleft palate, more marked on the left side, was also present. The tumor was readily removed under general anesthesia, and was reported to be composed of "fibrous tissue, unstriped muscle, etc."

A. Brown Kelly[31] describes a teratoid growth on a hairy polypus of the rhinopharynx in a 6-weeks-old female infant. The tongue-like growth was first noticed when the baby was one week old, and had been getting bigger and more troublesome. It was more than 2 inches in length and ½ inch wide in its thickest lower part. It was attached by a slender pedicle to the left side of the rhinopharynx, and frequently protruded from the mouth. The soft, fleshy body was covered with skin, stratified squamous epithelium, provided with numerous fine hairs; the underlying connective tissue was of a loose rudimentary character, with two or three minute glands; the central and larger portion of the polypus was composed of tissue closely resembling fat.

Greig[32] reports an instance of congenital Dupuytren's contraction of the fingers. This was observed in a well developed male infant, aged 5 weeks. The mother had noticed since birth a difficulty in getting the child's palms washed, in that she was unable to get the fingers extended. On careful examination under general anesthesia the condition was apparently not a tendinous affection, and there was no evidence of any abnormality in the growth, or peculiarity in the innervation of the muscle. The lesion appeared to involve the skin and the subcutaneous and deep fascia, and to be similar to the condition known as Dupuytren's contraction in adults.

Influenza.—During the recent pandemic of influenza the occurrence of a form of severe croup closely resembling clinically a true diphtheritic croup, but without the presence of the Klebs-Loeffler bacillus, was noted by a number of writers. Coray[33] relates that during the severe wave of influenza at Zurich many children developed laryngeal stenosis, and in one half of his eighteen cases emergency treatment was required. The resemblance to true diphtheria was so deceptive that it seemed antitoxin should be given as a routine measure in such cases, although it probably had no influence on the influenza. Coray was unable to find any mention of croup with influenza in the records of the 1890-1891 epidemic.

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31. Kelly, A. Brown: Teratoid Growth on Hairy Polypus of the Nasopharynx, Brit. J. Child. Dis. **15**:226, 1918.

32. Greig, D. M.: Two Cases of Unusual Congenital Abnormalities, Edinburgh M. J. **11**:384, 1917.

33. Coray: Ueber Kruppartige Affektionen bei Influenza, Cor.-Bl. f. schweiz. Aerzte **49**:475, 1919.

Regan[34] reports a series of twenty cases, five of which terminated fatally. The bacterial flora found by him was similar to that generally found among influenza cases. There was absence of the Klebs-Loeffler bacillus. The most effective treatment was the use of steam inhalations, followed by intubation, if necessary. The lesion as seen postmortem, is thus described by him:

Microscopic sections of the larynx and trachea show the surface epithelium of the mucosa to be intact, and without presence of fibrin formation, showing thus dissimilarity to diphtheritic laryngitis. The blood vessels of the mucosa and submucosa are seen to be markedly dilated. There is excessive exudation of polymorphonuclear cells, lymphocytes and serum into the surrounding tissues. The thickness of the mucosa and submucosa is greatly increased by this infiltration of tissues and dilatation of blood vessels.

V. B. Philpot[35] reports an instance of lymphangioma of the chest in a girl 4 years of age, with complete cure by operation. The child was born with two small tumefactions, one below the axilla and one just above the left clavicle. After a time these began to grow rapidly and the patient became weak and emaciated. The occurrence of abnormally high temperature, from time to time, was supposed to be due to some inflammation in the tumor. At operation it was found that a sac extended below the ribs and formed pockets all through the upper left chest, one of them extending to the scapula behind, and another between the clavicle and the scapula forming the large cyst to be seen above the left clavicle. Drainage, repeated curretting and swabbing effected a complete cure.

Canelli[36] has studied the size of the thyroid in infants prematurely born or dying a few days after birth. Assuming that the maximum weight of the normal thyroid is 4.85 gm., he found the thyroid enlarged in four of the seventy cadavers examined. In one instance the thyroid weighed 21.5 gm. No trace of iodin was found in the smallest of the four enlarged thyroids, but the largest one, 0.007015 gm. of iodin was present. A brief historical review of the subject is embodied in the paper.

Brennemann[37] reports four additional cases of congenital atresia of the esophagus seen by him since his report of three cases in 1913. In his experience no other congenital anomaly of the intestinal tract has

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34. Regan, J. C., and Regan, C.: Influenzal Croup, Am. J. Dis. Child. **17**:376 (June) 1919.

35. Philpot, V. B.: A Rare Surgical Case, South. M. J. **12**:147, 1919.

36. Canelli, A. F.: Contributo allo studio dello struma tiroideo congenito, Pediatria **27**:264, 1919.

37. Brennemann, Joseph: Congenital Atresia of the Esophagus, with Report of Four Additional Cases, with Three Necropsies, Am. J. Dis. Child. **16**:143 (August) 1918.

approximated even remotely such frequent appearance, with the possible exception of hyperplastic stenosis of the pylorus. In all of them the upper end of the esophagus ended blindly, the lower end connecting the stomach with the trachea. There is a striking lack of uniformity in the frequency with which this anomaly is met by pediatricians. Brennemann finds the symptomatology strikingly uniform.

Hymmson and Kahn[38] analyzed the intestinal contents (meconium) of five newly born infants. They found the iron and calcium contents similar to those found in hunger feces; the phosphorus was less, the sulphur was much increased. Traces of ammonia were found, but no uric acid. Extremely faint traces of amylose were found, but no trypsin, erepsin, lactase or lipase.

Batchelor[39] reports in greater detail the findings in her investigation of the dejecta of seventy-four children with special reference to the isolation of aerobic sporebearers. She found *Bacillus cereus* in thirty-two cases; *B. albolactus* in seventeen cases; *B. pseudotetanus* in nine cases; *B. mesentericus* and *B. subtilis* each in six cases; *B. petasites* and *B. vulgaris* each in two cases.

The normal occurrence of these spore bearing organisms and of yeasts in the intestinal contents is worthy of note, as is also their relation to the bacterial flora of the diet, and their susceptibility to change with changes in the diet,[40] as shown by Herter and Kendall, Torrey and others.

Lowenburg[41] reports a case of congenital unilocular cyst of the liver (hemangioma) in a male infant 19 months old. These cases are rare; they have been considered by Still (1898), Morris (1913) and Moschowitz (monograph). According to Moschowitz, operative results have been satisfactory. In Lowenburg's case the walls of the cavity were stitched to the abdominal wall, and the cavity was packed with gauze. The walls of the cyst were 1 inch thick, friable, brown, and suggested a membranous lining. The child reacted well after the operation. Later, however, there developed great drainage of serum from the cyst wall, entailing the loss of much water, and mineral and albuminous matter. The child gradually weakened, became asthenic (demineralized) and died.

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38. Hymmson, A., and Kahn, M.: Study of the Intestinal Contents of Newly Born Infants, Am. J. Dis. Child. **17**:112 (February) 1919.

39. Batchelor, M. D.: Aerobic Spore Bearing Bacteria in the Intestinal Tract of Children, J. Bacteriol. **4**:23, 1919.

40. Torrey, J. C.: The Regulation of the Intestinal Flora of Dogs Through Diet, J. M. Res. **39**:415, 1919.

41. Lowenburg, H.: Liver (Hemangioma) Congenital Unilocular Cyst of, Arch. Pediat. **35**:285, 1918.

Young and Wright[42] report an instance of congenital defect of the abdominal wall.

Carr[43] reports an instance of Hirschsprung's disease, in which secondary calcification of the lower part of the sigmoid and the upper part of the rectum had occurred. The patient, a girl, aged 6 years, was brought to the hospital in a condition of shock and died soon afterward.

Bacigalupo[44] describes a mucodermoid cyst of the pericardium. He considers that intrathoracic congenital tumors are rare and states that Nandrot[45] recorded the fifty-seventh case. In certain cases the cyst had extended to and become adherent to the pericardium, but in no case has the cyst involved the pericardium. Bacigalupo's case is believed to be the first case of congenital cyst of the pericardium to be described. The age of the patient is not stated.

Bronson and Sutherland[46] report an instance of ruptured aortic aneurysm in a child 5 years and 10 months of age. The lesion was a fusiform aneurysm of the secondary arch of the thoracic aorta. The aneurysm apparently followed a partial stenosis of the aorta between the insertion of the ductus arteriosus and the left subclavian artery. That this structure was a congenital anomaly was supported by the presence of a diaphragmatic hernia, subluxation of the joints, and defective cranial development.

The authors give a brief discussion of the etiology of aneurysms in children. From the literature they summarize two instances of aneurysmal dilatation due to partial stenosis of the thoracic aorta,[47] and also five cases of aneurysm apparently due to congenital anomalies in the structure of the ductus arteriosus; in two of the latter, rupture caused death. In the literature they find seven reports of death following rupture of thoracic aortic aneurysms in children, one case of rupture of the aorta without aneurysm, and two cases of rupture of aneurysms of the abdominal aorta. A roentgenogram and photographs of the specimen accompany the description of their case.

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42. Young, W. A., and Wright, E. J.: Congenital Defect of Abdominal Wall, J. Trop. M. **21**:158, 1918.

43. Carr, W. L.: Case of Hirschsprung's Disease, Tr. Am. Pediat. Soc. **30**:108, 1918.

44. Bacigalupo, Juan: Mucodermoid Cyst of the Pericardium, J. A. M. A. **71**:961 (Sept. 21) 1918.

45. Contribution a l'etude des kistes dermoids du mediastin anterieur, Thèse de Paris, 1917.

46. Bronson, E., and Sutherland, G. A.: Ruptured Aortic Aneurysms in Childhood, with the Report of a Case, Brit. J. Child. Dis. **15**:241, 1918.

47. Smith and Targett: Aneurysm of Aorta in a Boy Nine Years Old, Tr. Path. Soc., Lond. **48**:53, 1897. Wasastjerna, E.: Fall von Aortaruptur nach Schlittschulaufen, Ztschr. f. Klin. Med. **49**:405, 1903.

Rivers[48] describes an instance of erosion of the blood vessels of the neck in the course of scarlet fever. The patient was 5½ years of age. The symptoms at onset were not characteristic; albuminuria subsequently appeared, and on the twenty-third day after onset a characteristic scarlet fever desquamation was present. At the same time, the child became very ill, and a necrosis of the right tonsil, with a very foul secretion, was found. Two ounces of pus, but no blood, were evacuated from a mass the size of an egg on the right side of the neck. During the night the child was found exsanguinated, lying in a pool of blood. Physiologic sodium chlorid solution was given subcutaneously and a transfusion of mother's blood was done. An attempt was then made to examine the abscess cavity; there was a gush of blood, apparently from the external carotid or the lingual artery. These vessels were ligated and the hemorrhage was controlled. Another transfusion was given and the child improved for two weeks, when there was a second hemorrhage, the result of sloughing of the suture on the external carotid. The child then went on to recovery, though convalescence was complicated by a psychosis.

Howland[49] reports several cases which ended in death, with marked evidence of circulatory failure, but with no changes other than great cardiac hypertrophy and more or less dilatation. Postmortem the hypertrophy was the striking feature. None of the conditions in which hypertrophy usually occurs were present in any of these cases. The myocardium was normal; the musculature was intact. Howland thinks there is reason for believing that as a result of some nervous or muscular disturbance incoordinate action of the heart results.

Marriott and Howland[50] report experiments showing that the presence of acid phosphate in the body, even in the absence of renal disease, gives rise to the excretion of urine of a character such as has been previously observed only in nephritic acidosis. It is their opinion that the results of their experiments give additional confirmation to the view that the acidosis occurring in the course of nephritis is due to retention of acid phosphate.

Canelli[51] writes on congenital syphilis of the kidney. He states that this condition is found to be rather frequent at necropsy, being shown by the presence of sclerosis, atrophy, gummas and retention cysts.

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48. Rivers, T. M.: Hemorrhage Into a Postscarlatinal Cervical Abscess. Ligation of the Common Carotid. Recovery, Johns Hopkins Hosp. Bull. **30**:240, 1919.

49. Howland, John: Tr. Am. Pediat. Soc. **30**:2, 1918; abstr. J. A. M. A. **71**:221 (July 20) 1918.

50. Marriott, W. McK., and Howland, John: The Influence of Acid Phosphate on the Elimination of Ammonia in the Urine, Abstr. Bull. Johns Hopkins Hosp. **29**:264, 1918.

51. Canelli, A. F.: Sifilide renale congenita, La Pediatria **26**:257, 1918.

Amyloid degeneration of the kidneys is not characteristic of syphilis in the congenital form. There may be acute interstitial nephritis or a chronic sclerotic (atrophic) form. Tardy inherited syphilis is frequently manifested by a syphilitic albuminuria.

Hinman[52] reports a cytoscopic study of urologic conditions in children.

Comby[53] discusses the occurrence of suprarenal hemorrhage in children. Friderichsen[54] has shown that the condition presents a uniform and constant symptomatology and is not extremely rare, presenting three cases of his own and twenty-five from the literature. The attack may come on at any age. It occurs suddenly. The child becomes pale or cyanotic, and then quickly moribund, without noteworthy dyspnea or other signs of pulmonary affection. Necropsy always shows the same findings: cutaneous hemorrhages and hemorrhages into the cortex and medulla of both suprarenals. Comby presents three cases, occurring in children 6 months, 10 months and 14 years of age, respectively.

Comby[55] also presents a case of Addison's disease in a girl 13 years of age. The patient had probably been infected by her father, who had died of pulmonary tuberculosis. She presented all the symptoms of Addison's disease, including bronzing of the skin, profound asthenia, arterial hypotension and vomiting. Death occurred after an illness of about two months. The only lesions found at the necropsy were in the suprarenals. The right gland was converted into an abscess; the left gland was studded with caseous nodules.

Rutelli[56] describes another case of Addison's disease in a girl, 10 years of age. There was a history of enlarged cervical glands at the age of 10 months, and an affection of one finger which resulted in ankylosis at the age of 2 years. Pigmentation of the skin commenced at the age of 4 years. The blood pressure ranged from 65 to 70 mm., and there was marked asthenia; the urine was normal, except that there was slight diminution in the elimination of urea and creatinin. On a diet rich in carbohydrates, sugar appeared in the urine twenty hours after injection of 1 mg. of epinephrin; on an ordinary diet, after eighteen hours, and on a diet poor in carbohydrates, after four hours.

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52. Hinman, F.: Cytoscopic Study of Urological Conditions in Children, Am. J. Dis. Child. **17**:306 (May) 1919.

53. Comby, J.: Apoplexie surrenale chez les enfants, Arch. de méd. d. enf. **21**:651, 1918.

54. Friderichsen, C.: Hemorrhage in Suprarenals, Ugesk. f. Læger **79**:1817, 1917.

55. Comby, J.: Maladie d'Addison chez une fille de trieze ans. Mort. Autopsie, Arch. de méd. des enf. **20**:28, 1917.

56. Rutelli, G.: Contributo allo studio della matellia di Addison nell infanzia, La Pediatra **24**:274, 1916.

Control experiments in a healthy subject gave similar findings in twelve, eight and two hours, respectively.

Strick[57] reports a case of congenital absence of the uterus complicated with anomalous vulvovaginal anus. He presents statistical data on the occurrence of the condition.

Comby[58] discusses the occurrence of abscess of the anus in children. A case seen by him, the patient being 13 months old, recalls to mind an early series of seven cases, and he enumerates the causes of the condition. It usually appears in infants and is rarely tuberculous in origin.

The occurence of a pouch in the buttocks with an opening into the rectum is reported.[59]

Hayd[60] reports the occurence of a small round cell sarcoma in the right ovary of an infant, aged 23 months. It was the size of a goose egg, and was felt in the right lower quadrant as a large, movable, smooth tumor, not painful to the touch. Two or three weeks later the mass had become tender, and considerable fluid was present in the abdomen. On exploration, the tumor was found to be free in the abdomen, with the right tube attached to it. Bloody fluid was present; and on palpating the liver it was found to be much enlarged and filled with nodular masses. The child died several weeks later.

Mandelbaum,[61] in a study of two cases of Gaucher's disease in adults, offers the following conclusions: "Gaucher's disease is characterized by a distinctive, well defined clinical picture and constant, definite changes in the hematopoietic organs. The presence in these organs of peculiar large cells, with a characteristic type of cytoplasm, not duplicated in any other disease, is a distinguishing histologic feature. These cells are derived from the reticular apparatus of the hematopoietic structures, but an additional origin from the endothelial cells of the venous sinuses of the spleen cannot be denied. Fat or lipoid bodies cannot be found in the large cells by microchemical or polariscopic tests. The peculiar substance in the large cells does not lie in the extractive group, on chemical analysis, but is apparently of complex protein nature, in combination with lipoids. The disease is evidently caused by some disturbance of metabolism, the products being found in a specific group of cells (reticulo-endothelial) of the hematopoietic system."

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57. Strick, E. J.: A Case of Congenital Absence of the Uterus with Anomalous Vulvovaginal Anus, Am. J. M. Sc. **156**:75, 1918.

58. Comby, J.: Abses a l'anus chez les jeunnes enfants, Arch. de méd de enf. **21**:365, 1918.

59. Pouch in Buttocks Opening Into Rectum, Arch. Espanola de Pediat. **2**:533, 1918.

60. Hayd, H. E.: Sarcoma in the Left Ovary in a Child Twenty-Three Months Old, Am. J. Obst. **78**:764, 1918.

61. Mandelbaum, F. S.: Two Cases of Gaucher's Disease in Adults. A Study of the Histopathology, Biology and Chemical Findings, Am. J. M. Sc. **157**: 388, 1919.

Carr and Moorhead[62] report a case of Gaucher type of splenomegaly.

Inaba and Ohashi[63] report the finding of a new microbody in the blood in a case of myelogenous leukemia.

Jemma[64] reports a case of malignant lymphogranuloma in a girl, 6 years old. The disease had existed for ten months. The tumors were situated chiefly in the cervical region. There was intermittent fever and increasing pallor and weakness. The von Pirquet test was positive. The blood examination showed 53 per cent. lymphocytes—in a total count of 7,600 leukocytes—and tubercle bacilli. Scattered Schrön-Much granules were found in material taken from the glands.

Ward[65] discusses the nature of von Jaksch's disease.

Schwarz[66] discusses the occurrence of a chlorotic type of anemia in infants and children.

Wolbach and Morse[67] report a case of neuroblastoma sympaticum, and Humbort and Naville[68] report a case of neurofibromatosis.

Barron[69] reviews the subject of meningitis in the newly born and in early infancy. He refers to Koplik's recent paper[70] and tabulates forty-two cases reported in the literature since 1895, when Sherer appears to have reported the first cases. The causative organisms in these cases were, in order of frequency: *B. coli,* fourteen cases; staphylococcus and streptococcus, six cases; meningococcus cases, five; streptococcus, four cases; pneumococcus, 4 cases; tuberculosis, three cases, and *B. lactis-aerogenes, B. pyocyaneus, B, mucosus-capsulatus, B. typhosus, B. influenzae, B. catarrhalis,* one case each. In Barron's case—a male infant, born in breech presentation in which the delivery was assisted by inserting a finger into the mouth and then exerting a moderate amount of traction, and which died from meningitis on the eleventh day—the causative organism was definitely *B. coli-communis.*

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62. Carr, J. G., and Moorhead, L. D.: Report of a Case of Gaucher Type of Splenomegaly, J. A. M. A. **72**:19 (Jan. 4) 1919.

63. Inaba, Ttsuyoshi, and Ohashi, Shin: A New Microbody Found in the Blood in a Case of Myelogenous Leukemia, etc., Am. J. Dis. Child. **16**:1 (July) 1918.

64. Jemma, R.: Sul linfogranuloma maligno, Pediatria **26**:513, 1918.

65. Ward, G.: Nature of von Jaksch's Disease, Brit. J. Child. Dis. **15**:101, 1918.

66. Schwarz, Herman: The Chlorotic Type of Anemia in Infants and Children, Arch. Pediat. **35**:430, 1918.

67. Wolbach, S. B., and Morse, J. L.: Neuroblastoma Sympaticum, Am. J. Dis. Child. **16**:63 (July) 1918.

68. Humbort, G., and Naville, F.: Neurofibromatosis, Ann. de méd., Par. **5**:108, 1918.

69. Barron, Moses: Meningitis in the New-Born and in Early Infancy, Am. J. M. Sc. **156**:358, 1918.

70. Koplik, H.: Meningitis of the New-Born and in Infants Under Three Months of Age, Arch. Pediat. **33**:481, 1916.

Barron tabulates also the probable portal of entry in nineteen of the forty-two cases. The figures are as follows: spina bifida, four cases; otitis media, four cases; hematogenous, three cases; eustachian tube, one case; mouth, one case (Barron's case); lungs, umbilicus, intestine, circumcision wound and placenta, each one case. Meningitis in the new-born and in early infancy is a rare disease.

It appears that *B. coli* occupies in the meningitis of early infancy the important place held by *B. tuberculosis* in the later months of infant's life. The pathogenic strains of *B. coli* may show marked variations in form, in rates of fermentation and in motility. Most of the strains isolated in the cases tabulated by Barron were actively motile. The avenues of infection in the new-born, in general, have not been definitely established. La Fetra[71] believes that infants are born septic very rarely. Scherer, Noeggerath and Hermann believe that contaminated water of the bath tub is a frequent source of infection. According to Scherer, infected liquor amnii furnishes a similar possibility. Aschoff,[72] in the course of careful studies, found that the passage of liquor amnii up the eustachian tubes during premature respiration is fairly common; he believed that resulting inflammation occurred not from infection thus introduced, but only from the fluid acting as a foreign body, a locus minoris resistentiae being caused. Bonhoff and Esch[72] held that infection occurred in this manner in their case. Rasch[72a] believes that infection of the external auditory canal is more important than infection of the eustachian tube. Others believe that the umbilicus is the most important route. The possibility of injury of the mucous membrane of the mouth by the accoucheur's finger giving a portal of entry has been suggested. Barron thinks this occurred in his case. Infection through the intestinal tract is possible.

The susceptibility of infants to infection with organisms that are otherwise only slightly pathogenic may be explained, concludes Barron, by the feebleness of antibody production during the early months of infancy (Halban and Landsteiner); and the greater resistance of breast fed infants over artificially fed ones is probably due to the compensation of the passive immunization by the breast milk for the active immunization which is still deficient.

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71. La Fetra, L. E.: Accidents and Diseases of Early Weeks, Arch. Pediat. **33**:401, 1916.

72. Bonhoff and Esch: Ueber einen Fall von Meningitis purulenta beim Neugeborenen, infolge rechtseitiger Mittelohrentzündung, Ztschr. f. Geburtsh. u. Gynäk. **70**:886, 1912. Cited from Barron.

72a. Rasch: Ueber die Hauflichkeit und Bedeutung von Mittelohrentzündung bei Kleinen Kranken Kindern, Jahrb. f. Kinderh. **37**:319, 1844. Cited by Barron.

Kramer and Wright[73] report an instance of meningitis due to *Streptococcus pyogenes-hemolyticus* in a mother and her unborn child. The fetus was 40 cm. long. The authors believe that this occurrence supports Rosenow's dictum of elective localization in the maternal and fetal meninges.

Hunt[74] reports a case of juvenile paralysis agitans.

Naville[75] reports the microscopic findings in a typical case of amaurotic family idiocy. The infant was the fourth in a family of six children to be thus affected. Naville describes aplasia of the tracts in addition to the usual lesions of this form of idiocy. Clinically, he recognizes a juvenile form and atypical forms, besides the usual infantile form. All are believed to proceed from the same pathognomonic degeneration of the cells. The eye findings may vary widely. A connection with certain other familial diseases of evolution is suggested, essential atrophy of the optic nerve, Marie's cerebellar ataxia, etc., and certain mendelian features are pointed out.

Talbot[76] reports studies on the energy metabolism of an amaurotic family idiot.

28 East Mount Vernon Place.

73. Kramer, G. B., and Wright, W. B., Jr.: Streptococcic Meningitis in the Mother and Child in Utero; Report of a Case, J. A. M. A. **71**:729 (Aug. 31) 1918.

74. Hunt, J. R.: Case of Juvenile Paralysis Agitans, Primary Atrophy of Pallidal System of Corpus Striatum, Neurol. Bull. **1**:237, 1918.

75. Naville: Pathological Anatomy of Amaurotic Familial Idiocy, Arch. f. Neurol. u. Psychiat. **1**:286, 1917.

76. Talbot, Fritz B.: Energy Metabolism of an Amaurotic Family Idiot, Am. J. Dis. Child. **16**:39 (July) 1918.

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